MUSCLE SPASM AND DEGENERATION IN INTRATHORACIC INFLAMMATIONS

THEIR IMPORTANCE AS DIAGNOSTIC AIDS AND THEIR INFLUENCE IN PRODUCING AND ALTERING THE WELL ESTABLISHED PHYSICAL SIGNS, ALSO A CONSIDERATION OF THEIR PART IN THE CAUSATION OF CHANGES IN THE BONY THORAX

AND

LIGHT TOUCH PALPATION

THE POSSIBILITY AND PRACTICABILITY OF DELIMITING NORMAL ORGANS AND DIAGNOSTICATING DISEASED CONDITIONS WITHIN THE CHEST AND ABDOMEN BY VERY LIGHT TOUCH

BY

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SIXTEEN ILLUSTRATIONS

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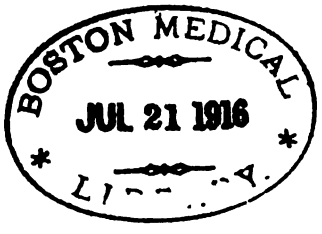

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TO THE MEMBERS OF THE MEDICAL PROFESSION WHO ARE EVER STRIVING TO SERVE THEIR FELLOWMEN BY MAKING EARLIER AND MORE ACCURATE DIAGNOSES, THIS BOOK IS DEDICATED BY

THE AUTHOR.

INTRODUCTION

The material in this monograph is, with a few changes, the same as that which appeared in Brauer's Beiträge zur Tuberkulose, Band XXII, Heft 1, 1912. It represents original observations made by the author during the past three years. The observations and practical suggestions here recorded are based on several thousand physical examinations. They have been carefully tested and checked by other methods of diagnosis. The author gives them to the medical profession in this form at the earnest solicitation of many of his colleagues, hoping that they may prove of value in furthering diagnosis, especially of intrathoracic diseases; and particularly hoping that the appreciation of the early changes in the muscles covering the apex and in the diaphragm may aid in the early diagnosis of tuberculosis.

Should the practical suggestions herein made fail to furnish the aid in clinical diagnosis for which the author hopes, he still believes that the theoretical considerations presented may be of value to the profession. The facts herein presented are not wholly new. Many of the principles and conditions have been previously described. The author only claims originality in that he has worked them out as an independent observer and has made application of them which had not heretofore been made. The importance of this work does not rest with that which is written here, but lies in the far-reaching effect which it may have on diagnosis as a science. If the condition of the muscles as I describe it, exists; if the influence of their changed condition is as great as it seems to be; and if light touch is sufficient to outline the deep borders of organs lying within the chest and abdomen, then many of our supposedly well-established theories in physical diagnosis are at fault and the entire subject must be rewritten.

Many facts and observations pertaining to medicine have been recorded during the past centuries which are little short of wonderful, when we consider the meager facilities for investigation. Few of the theories based on these observations, however, have wholly withstood the searchlight of modern methods, nor do we yet feel that all error has been eliminated; nevertheless we are entering upon a new century with a fund of knowledge which makes the practice of medicine more

satisfactory than ever before. There is still room, however, for doubting, questioning, and intelligent investigation. Physicians with experience must not be afraid to record observations which differ from so-called established facts. This is especially true of clinical observers. Clinicians were so hypnotized by the brilliant results of surgery and so impressed by the wonders of the laboratory that, for a time, they did not produce their share of new observations; and so, clinical medicine lagged behind the departments of experimental medicine and surgery. With the advent of specialization and its present-day development, however, clinical methods are coming more to the fore. Surgery is brilliant; the laboratory is attractive; but it is the clinician who must give the final word; so he should constantly be looking for new methods and new facts, and should cultivate a recipient, though discerning, mind, that he may carefully weigh new evidence laid before him.

The change on the part of the muscles and light touch palpation, as outlined in these pages, emphasizes inspection and palpation, which have been heretofore neglected in the diagnosis of intrathoracic diseases. When the principles herein described are thoroughly comprehended, the examiner will be able to obtain by inspection and palpation far more knowledge than can be obtained by percussion.

It is not expected that these will displace the older and tried methods, but it is hoped that they may supplement them, and aid the examiner in making a more accurate diagnosis.

It is earnestly hoped that these methods of diagnosis may prove as practical to the profession as a whole as they have to the author and that they may result in benefit to both physician and patient.

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MUSCLE SPASM AND DEGENERATION IN INTRATHORACIC INFLAMMATIONS

CHAPTER I

Spasm and Degeneration of the Muscles of the Neck and Thorax in the Presence of Intrathoracic Inflammations

Obtaining accurate information concerning the organs lying within the great cavities of the body is beset with many difficulties. These difficulties are: first, those depending upon the inaccuracy of our methods of obtaining information; and, second, upon the limitations of the examiner.

The data obtained by physical examination is derived, for the most part, by inspection, palpation, percussion, and auscultation. Inspection and palpation, if we may judge by the text books on physical diagnosis, are of limited value in examination of the chest, while the most valuable data are derived by percussion and auscultation. These methods, however, are difficult to master and require large experience and constant use in order to give reliable results. Consequently, there are comparatively few men who are masters of them. Palpation, percussion, and inspection are considered to be of great value in diagnosis within the abdomen, while auscultation is only of limited value.

During the past three years I have made certain observations and developed certain methods of examination which increase the relative importance of inspection and palpation, especially as applied to the chest, and show them to be of the greatest practical value in every-day practice. It is possible by these methods alone to determine more or less accurately whether or not disease is present in the chest, whether or not it is active or quiescent, and also as to its extent. Aside from new methods of examination being based upon these observations, they also aid in explaining many of the well-recognized signs and many of the phenomena derived by auscultation and percussion.

The superficial tisssues and the outlines of the chest of individuals whose intrathoracic organs are the seat of inflammatory lesions may show certain departures from the normal which are due to the lesion. Some of these changes have been described as predisposing to disease when in reality they are a result of it.

Diminished motion, contractions, irritability of the muscles on percussion, and the general wasting of the tissues have long been recognized in tuberculosis. The barrel-shaped chest of emphysema is well-known. Localized contractions of the chest wall often follow pleurisy and emphysema. Freund and his followers (1, 2, 3, 4, and 5) have called attention to ankylosis of the first costosternal articulation and shortening of the first rib as being predisposing causes of tuberculosis. Rothschild (6) regards a lessening of the manubrio sternal angle as a predisposing cause of the same. Head (7) has shown that certain areas in the skin covering the chest show trophic changes and are altered in their relations to pain, heat, and cold when the lung is involved. Others, among whom are White and Van Norman (8), have confirmed these findings. Mackenzie (9) calls attention to the spasm of the intercostal muscles which occurs in cases of angina pectoris. Musser (10) and West (11) mention spasm of the chest muscles overlying areas of acute pleurisy. Cyriax (12) described spasm of the chest muscles, particularly the intercostals, in intrathoracic inflammations and suggested this condition as one to be relieved by manipulation. He did not consider it, however, as far as I am aware, as a condition of diagnostic importance, at least not in his early paper. Jessen (13) mentions atrophy of the trapezius over the diseased apex.

Bálint (14) finds an altered electrical reaction on the part of the muscles on the side of pulmonary involvement. Boenniger (15) describes a unilateral lymph stasis affecting the muscles of the affected side in diseases of the lungs and pleura.

Coplin (16) made a pathological study of the intercostals and diaphragm as affected by direct extension of inflammation from the pleura and lungs.

In 1909 (17, 18, 19, 20, 21, and 22) I first described certain changes that I had noted in the muscles overlying inflammatory areas of the lungs and pleura and suggested this as a practical and valuable sign in physical diagnosis.

The conditions which I have noted are a spasm of the muscles of the neck and thorax on the affected side when the lungs or pleura are

acutely inflamed and a degeneration of the same when the process has passed over into a chronic state. Depending upon the acuteness of the inflammatory process affecting the pulmonary parenchyma and pleura, the muscles of the neck and those covering the chest show all gradations of change from the acute spasm to severe degeneration. Often the same thorax presents widely different conditions in different groups of muscles. These changes, while perceived by inspection and ordinary palpation, become especially noticeable when examined by light touch palpation.

My earlier descriptions were more or less incomplete and inaccurate. Because of my inability to fully grasp the condition as it presented itself, I could find no physiological basis which had been worked out to explain it. I could find no description of reflexes from the lungs and pleura affecting the motor nerves supplying the neck and chest muscles. All I had at my command was the clinical fact that the muscles are in spasm in the presence of acute inflammation and degenerated in the presence of chronic inflammation; and the anatomical fact that the intrathoracic viscera are supplied by the cervical sympathetic nerves and the superficial muscles are supplied by nerves having their origin in the same part of the cord as the sympathetics. Finally, I came to the conclusion that the motor changes must be analogous to those of sensation, which have been carefully worked out by Head, as mentioned above; and, while I have no schema which will show the path of the stimulus from the various portions of the lung and pleura to the cord and the distribution of the corresponding segmental reflexes to the muscles, yet I do not doubt that when this has been carefully worked out, it will offer the true explanation. It is probable that there may be some inaccuracies in my clinical observations which can be eliminated when the reflexes have been properly worked out; nevertheless, I believe that I have been able by clinical observations alone to work out valuable and fairly accurate diagnostic signs.

Aside from the changes in the musculature of the neck and thoracic wall, there is another phenomenon which has been heretofore described which has probably a similar explanation. I refer to the lessened motion of the diaphragm as described by Williams (23). This insufficient contraction as shown by a failure of the diaphragm to sink as low as it should on inspiration is now well-recognized in not only tuberculosis, as described by Williams, but also in pleurisy and pneu-

monia as described by de la Camp (24). De la Camp has suggested as a cause of this lessened action of the diaphragm the injury to the phrenic nerve on account of apical pleural adhesions. Hofbauer and Holzknecht (25) have suggested that it is due to a decreased elasticity in that portion of the lung that is involved in the tuberculous process and the retraction of the remaining tissue, causing a general lessening of the contractile power of the lung as a whole.

While these explanations seem plausible, yet our anatomical studies show that the phrenic nerve is given off from either the third and fourth or fourth and fifth cervical roots. Clinical observation shows that the superficial muscles which arise from this portion of the cord are thrown into spasm during acute inflammations in the lung and that they degenerate when the inflammation assumes a chronic form. From this it would seem that we are justified in attributing the same conditions to the diaphragm. In this opinion I am further strengthened by many other observations. Many phenomena can be traced directly to the nerves originating in the cervical portion of the cord when the lungs and pleura are the seat of acute or chronic inflammation. These phenomena show themselves in the skin, subcutaneous tissue, muscles, and joints.

Aside from the changes noted in the muscles of the neck and chest I would mention: the changes in the muscles of the arm which show themselves as both spasm and degeneration; a localized wasting of portions of the skin, and subcutaneous tissue which derive their nerve supply from the segments of the cervical cord; the changes in sensation as noted by Head; the trophic disturbances in the costosternal joints which result in chronic inflammatory change and ankylosis; dry arthritis, which affects particularly the shoulder joint; the vague aching pains which are so commonly felt in the interscapular region and about the shoulder joint in patients suffering from pulmonary tuberculosis, as well as the instance of regional neuritis which affects the nerves of the side involved.

When we recognize all these disturbances as being of segmental origin it strengthens us in our opinion that the diaphragm phenomenon, first described by Williams, is probably of the same segmental origin. In passing I would like to call attention to the pains which so often occur in the shoulder joint in patients who are suffering from tuberculosis. This is often diagnosed and treated as rheumatism, when in reality it is most probably due to an inflammation of the nerve result-

ing from the reflex segmental stimulations (47). In this connection Jessen has described a neuritis and perineuritis affecting these nerves. I have often seen the muscles of the arm on the side of a chronic involvement show a marked state of degeneration when compared with the muscles on the unaffected side. I would also recall that Bálint has described a change in the electrical reaction in these same muscles.

I find that one of the difficulties encountered by those who have attempted to elicit the changes which I have described is a failure to know the normal muscles and other soft tissues. Little study has been given to the superficial tissues covering the thorax. In examining the abdomen and the viscera contained therein for diseased conditions, the resistance of the muscles has always been noted; but, in examination of the chest, localized muscle spasm and degeneration have not long been known. One of the important principles in diagnosis is: that, in order to understand the abnormal, the examiner must familiarize himself with the normal. This principle is as important in connection with the muscles and superficial tissues of the chest as it is in any other part or organ.

What might be called a normal or well-formed thorax is probably much rarer than would seem at first thought. Such a thorax in an adult should be symmetrical on both sides. Beginning at the clavicle it should bulge forward, reaching the maximum point on a level with the third or fourth rib, and then gradually flatten out again as the lower border of the rib is reached. The supraclavicular and infraclavicular spaces should be well-filled and almost even with the clavicles themselves. The scapulæ should stand symmetrical. The ribs and intercostal spaces should be well-covered with subcutaneous tissue and muscles so that the intercostal spaces are barely recognizable in the upper two-thirds of the thorax, and are only seen distinctly in the lower portion where the musculature is thin. There should be a general symmetry in the muscles of the two sides, no individual or group of muscles standing out with undue prominence unless it be those that are increased in size by greater use than others, such as the deltoideus, trapezius, rhomboidei, and pectoralis in persons who do heavy work and use one hand far more than the other. The anterior neck muscles should not stand out unduly unless the patient is emaciated. Neither should the neck and chest muscles appear degenerated or atrophied under normal conditions.

There are certain conditions about the chest both as to its general

contour and as to the condition of the soft parts that are likely to lead to error unless they are understood. I have just mentioned that certain groups of muscles are often over-developed and appear larger on inspection and more resistant than normal on palpation, simply as a result of increased use. I would like to call attention to the fact that, if conditions should arise which would require less use for these muscles, they undergo retrograde change and present the appearance of degeneration. I have noted this degeneration from disuse in many instances where men who had been accustomed to perform such work as would call for the over-development of the arm muscles, particularly the pectorales, rhomboidei, trapezei, and deltoidei, were forced to change their occupations to those which would require the use of these muscles to a lesser extent. Under such conditions the muscles show degeneration and appear very much like the muscles I am describing as being degenerated because of the reflex stimulation resulting from inflammation of intrathoracic origin. The general atrophy accompanying old age should also be remembered.

Another condition which has puzzled me considerably, and one which I am not able to explain entirely satisfactorily, is a sinking of the supraclavicular notch which I have found to occur very commonly in adults. It has seemed to me to be most probably due to occupational influences, and yet I have not been able to satisfy myself definitely on this point. My reason for assigning it to this cause is the fact that it is most commonly found affecting the right side and seems to be more common in men than in women. This condition gives about the same appearance as that which accompanies an old lesion at the apex and which is often described as a contracted apex. The clavicle is prominent and the supraclavicular and infraclavicular notches are both deep, yet from physical examination in a portion of these cases I have not been able to satisfy myself that there has been a previous tuberculous lesion. In some patients who were right-handed, and who had an old lesion at the left apex, I found the left supraclavicular and infraclavicular notches prominent and the shoulder muscles flattened and more degenerated than on the other side, which suggests that infection and not occupation is the cause. This condition requires further study.

It is well to bear these conditions in mind in order to avoid error in diagnosis. It must also be remembered, however, that very often the same apex that shows these phenomena may be the seat of a tuberculous

lesion; but this can usually be determined by the careful application of palpation, percussion, and auscultation.

The normal condition of the child's chest differs widely from that of the adult. At birth, the first rib is on a level with the first dorsal vertebra (as shown in Fig. 1a). The anterior wall of the thorax gradually sinks so that by the time adult life has been reached, the first rib is on a level with the third dorsal vertebra (as shown in

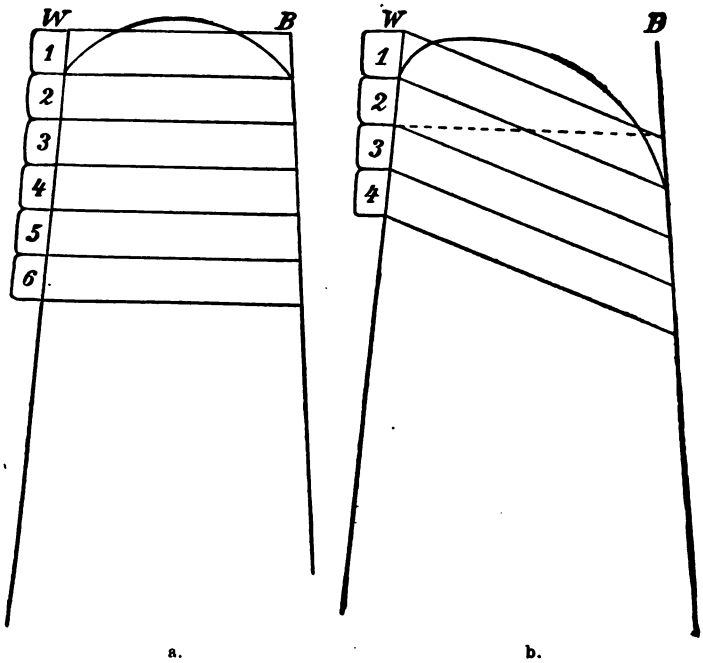

Fig. 1.—Showing schematically, the difference in elevation of the anterior portions of the ribs in children and adults. This shows plainly that the pull of the scaleni and sternocleidomastoidei muscles when in contraction, being attached to the first and second ribs and the sternum and clavicle anteriorly and to the cervical vertebræ posteriorly, affords a greater compressing force in early life than in later life.

Fig. 1 b). This sinking is probably due, at least in part, to the assumption of the erect posture; but it takes place gradually and does not reach its maximum until after puberty. This must be borne in mind in studying the effect of muscle change on the thorax of children.

Normal muscles have a certain elastic feeling common to all; the consistency of the muscles, however, differs for different individuals and for different groups of muscles. There is also a marked difference between the muscles of children and adults. The examiner should familiarize himself with the feeling of the normal muscles so that he may be able to recognize any departure from it.

Preliminary to making an examination of the muscles of the neck

and thorax for either spasm or degeneration, it is especially important to have the patient sit comfortably with all muscles relaxed. Personally, I prefer to have the patient sit erect, rather than recline. The head should be held in its natural forward position, and the arms should be allowed to hang loosely at the side. It is a mistake to tell the patient to hold his head up and look straight ahead. This will defeat the purpose by putting certain muscles on the stretch. What is wanted is for the patient to assume a natural relaxed position, which will usually be with the chin dropped on the chest, and usually with the head bent to one or the other side. There is the least tension on the shoulder muscles when the arms are resting in the lap. These precautions I deem absolutely essential; for, by a slight turn or twist of the head or arm, the neck and shoulder muscles are put on tension and incorrect impressions are gained. It might be thought that the ease with which these muscles are put on tension would vitiate the value of the sign, but not so, for it requires only a little care on the part of the examiner to secure complete relaxation. I have found it much more difficult to make out the muscle change when the patient is lying down than when sitting up.

It is also important to have the patient breathe quietly when palpating the chest muscles, because deep breathing causes the muscles of respiration to be contracted and makes them assume a degree of firmness which stimulates the condition of pathological reflex spasm for which we are examining.

Very commonly errors are made on percussion as well as inspection and palpation by disregarding the condition of the neck muscles. Often the examiner turns the patient's head to the opposite side while percussing one apex and fails to require the same relative position while percussing the other one. Realizing that contracted and relaxed muscles give different percussion results and that different positions of the head cause contraction and relaxation of different muscles, the importance of examining both apices under the same relative muscle condition becomes self-evident.

We will illustrate muscle changes by discussing them in relation to our most common apical disease, tuberculosis. A study of figures 2, 3, and 4 will show the important superficial muscles. Upon examination of a patient suffering from an active primary tuberculous lesion of one apex, the muscles present the following conditions: on inspection anteriorly, the sternocleidomastoideus, either the sternal or

clavicular portion or both, and quite often the scaleni, will be seen to stand out more prominently than on the other side. At times, the side of the neck on which the involvement is found near the clavicle, appears markedly uneven, compared with the other side, because of these mus-

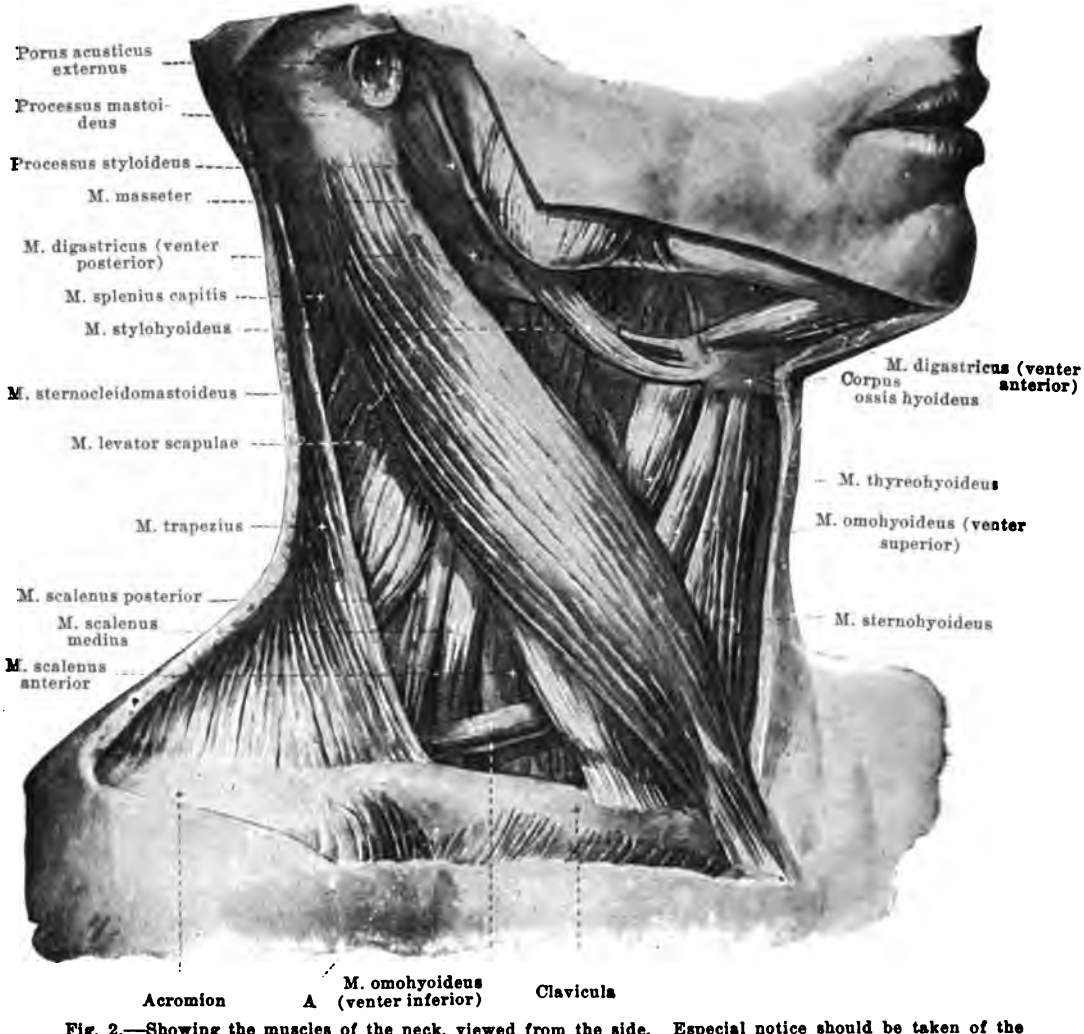

Fig. 2.—Showing the muscles of the neck, viewed from the side. Especial notice should be taken of the sternocleidomastoidei and scaleni. The space between the clavicular portions of the deltoid and pectoralis should also be noted.

cles standing up in distinct ridges. The belly of the sternocleidomastoideus is also sometimes much larger than normal giving the neck on the side of the involvement a full appearance. In one of my cases this was so marked that after the disease had healed and the muscles had degenerated the patient wore a collar of smaller size.

Viewing a patient with a primary involvement of the apex, posteriorly we often note a prominence of the trapezius both in the shoulder and cervical portions. The suprascapular notch often appears fuller than normal. This often affects the levator anguli scapulæ also and if

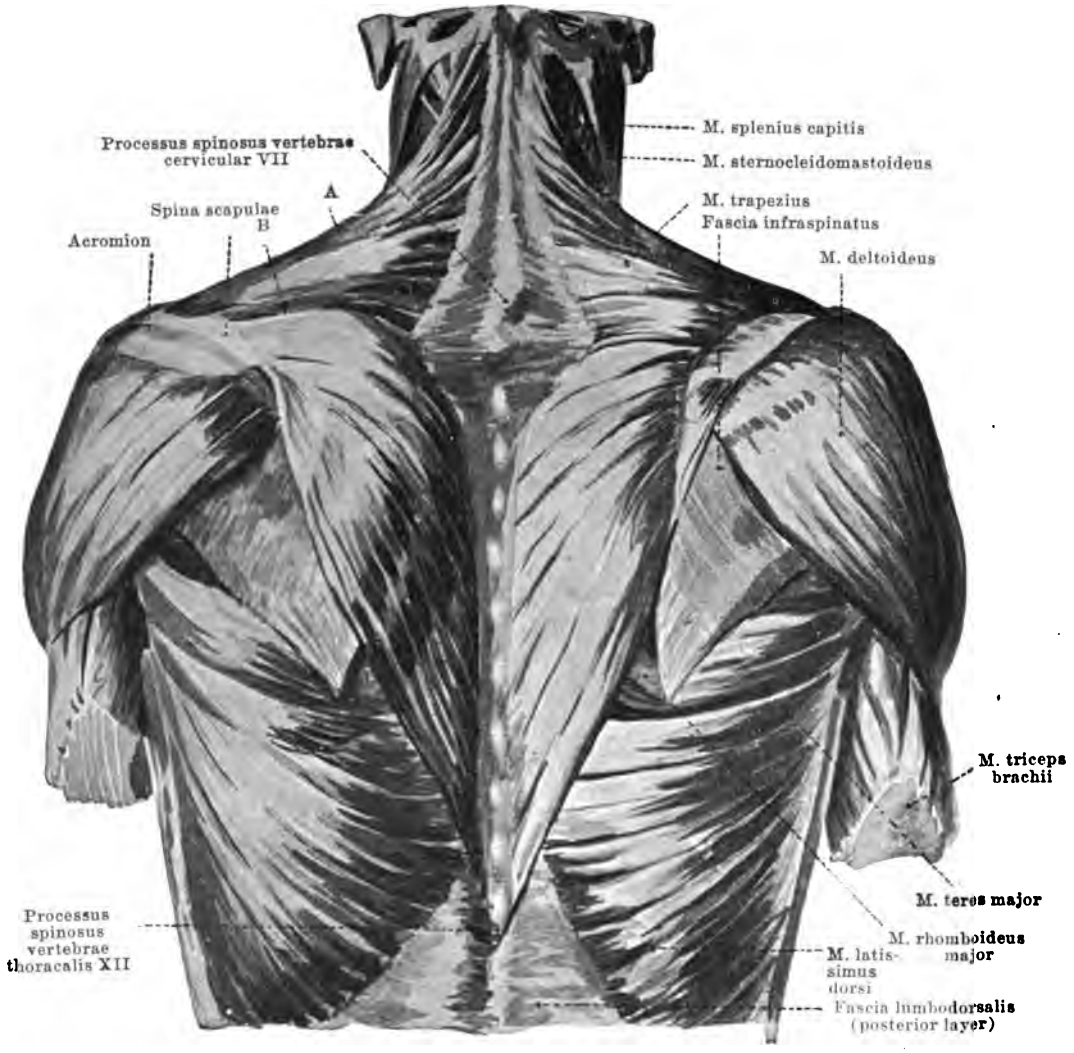

Fig. 3.—Showing the muscles of the neck posteriorly and the broad muscles of the back. The shoulder portion of the trapezius (A) usually shows spasm plainest, while degeneration often shows as a flattening or cupping of that portion immediately above the spine of the scapula (B).

the disease extends a little lower the rhomboidei likewise show spasm. On palpation these muscles feel decidedly firmer than normal.

I shall later take up the muscle condition in the presence of chronic tuberculosis, but I must mention here the condition which presents it-

self when renewed activity is found in an old chronic or quiescent lesion. If there has been an old chronic lesion at the apex, extending down over the anterior portion of the chest, we find the skin, subcutaneous tissue and the muscles all in a state of degeneration; the pectoral presents

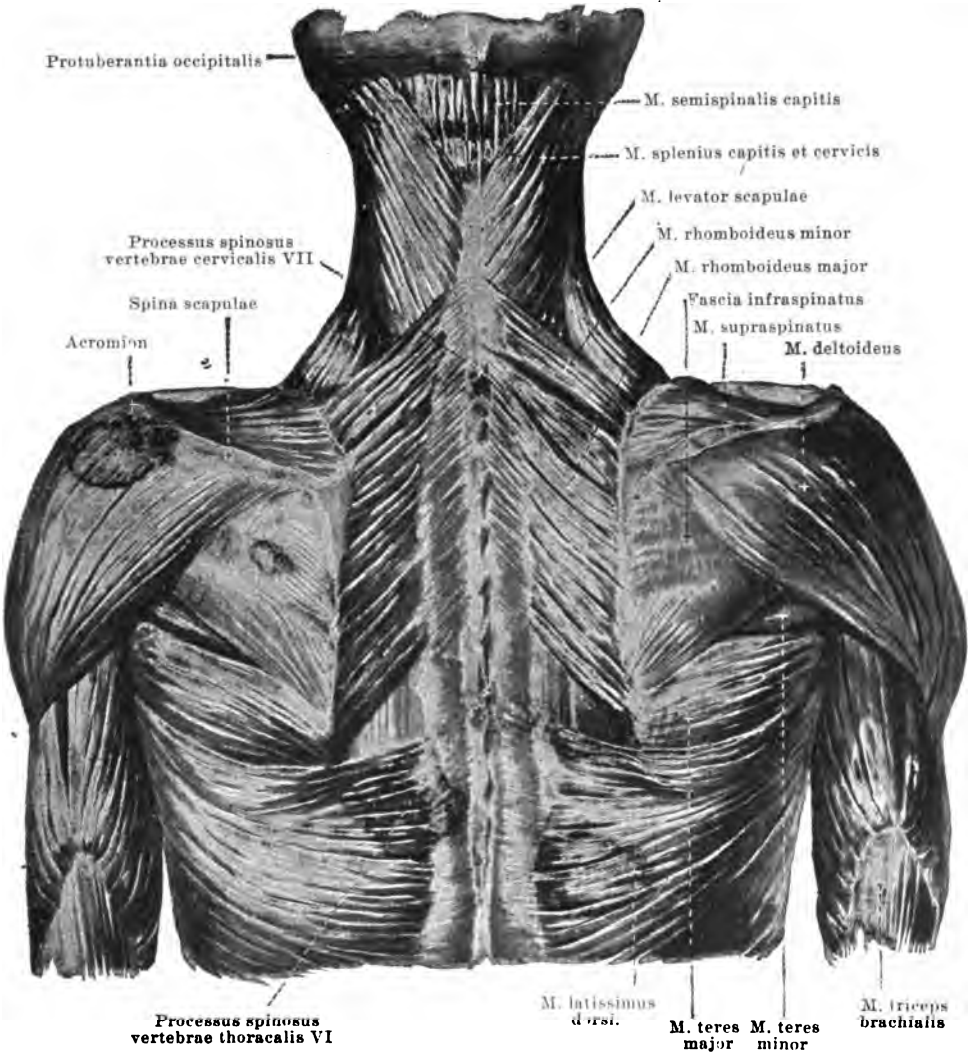

Fig. 4.—Showing the second layer of muscles of the back. Those of special interest in diagnosis are the levator anguli scapulae and rhomboidei.

a doughy feeling on palpation and as a rule its bundles are easily separated. The skin can be lifted up readily owing to the wasting of the subcutaneous tissue. The mamma itself is often atrophied and appears smaller than on the unaffected side. The neck muscles are also degenerated and smaller than usual. This shows itself particu-

larly in the sternocleidomastoideus; but, there is more than degeneration present. The activity in the lung has once more set up an irritation and produces a spasm of these degenerated muscles, which shows itself in a feeble contraction; so under these conditions we have both degeneration and spasm present.

Posteriorly, the same general conditions are present. The trapezius, which, when in spasm in primary lesions, often causes the suprascapular fossa to be fuller than normal, when degenerated often causes it to appear flattened or cupped. In rolling the muscles under the fingers the bundles separate very easily. The same degenerated condition is noted in the levator anguli scapulæ. The cervical portion of the trapezius appears smaller than normal. Aside from this degeneration the muscles are in contraction the same as mentioned in the anterior portion of the chest.

It will thus be seen that muscles which are in spasm, likewise those which show degeneration, differ very markedly from normal. When in spasm they are firmer to the touch and give one the distinct feeling of increased tension. Often the more tendonous parts of the muscles, such as the heads of the sternocleidomastoidei and scaleni, feel like distinct cords, while the more fleshy parts of the muscles, like the trapezius, levator anguli scapulæ, and the body of the sternocleidomastoidei, show their spasm by being larger than normal and by showing a firmer feeling than normal. When degeneration occurs there is a loss of the substance between the bundles, the subcutaneous tissue covering the muscles wastes away, and the muscles themselves lose their elasticity and feel doughy to the palpating finger.

If the disease is on the anterior surface of the lung, the anterior muscles seem to show greater change than the posterior muscles, and vice versa. When the entire apex is involved, all muscles covering the apex seem to show the spasm. When the lesion is very small or when the patient's neck is full, the muscle spasm, though present, may not be detected on inspection. When both apices are involved the change may be observed on both sides and for the want of comparison may, at first, be confusing; but the increased resistance will usually be made out after careful examination.

If the disease becomes more extensive and spreads downward, the muscle change extends with it. When it extends beyond the clavicle the intercostales, as well as the superficial muscles, show the change. This is well illustrated by the rigidity of the pectoral muscle when an

acute process extends down to the fourth or fifth rib before the patient has become emaciated. By picking up the anterior folds of the axilla one notices the same resistance on the part of the pectoralis as is noticed when it is in voluntary contraction. The edge of the muscle is sharp and unyielding. The same may be noted when the pleura covering the same portion of the lung is acutely inflamed.

From a scientific rather than a practical point of view, we must bear in mind that it might be possible for an inflammation in one lung to produce an irritation of the nerve fibers supplying the muscles on the other side as well as the side of involvement, and in one or two instances I have thought that this probably occurred in cases that I have seen, for I could not determine any other signs on the one side, yet it is just as natural to suppose that a lesion might have been present but overlooked. Head finds the same to occur, I believe, in his areas of sensation. However, this is so rare that it can be disregarded in practice.

In palpating the muscles it is well to use both light and moderately heavy touch. I often find, however, that I can detect a change in the fleshy muscles easier by light than by heavy touch. When the superficial muscles can be taken up and rolled between the thumb and fingers, it is well to examine them in this way. For examining the intercostales, the instructions given in my former paper should be followed (18). "The palpation is best done by gently pressing the tips of the fingers into the intercostal spaces and moving the hand sideways while noting the degree of resistance. During this procedure the fingers should not be allowed to slip on the skin for it is the condition of the muscles and not the skin that is noted."

After the inflammatory process in the lungs or pleura has existed for some time and passed over into a chronic state, the condition of the muscles change. They become degenerated and their bundles are easily separated. To the palpating finger they appear doughy. Their normal elasticity is gone. Coincident with this change in the muscles there is a thinning of the skin and a disappearance of the subcutaneous tissue. Some of these degenerative changes are evident to sight as well as to touch. Wheaton (26), noting this atrophy of the skin, described it as a sign of diagnostic importance in early tuberculosis. On the contrary it is a part of the general degeneration which occurs in the skin, subcutaneous tissue, and muscles and occurs after the process has existed for some time. It denotes chronicity rather than earliness

although it is often found over a comparatively early tuberculous process. In explanation of this, however, we must remember that many patients coming to us for the first time, suffering from a slight lesion, present what is usually considered to be an initial involvement, when, in reality, it is an old quiescent focus which has become the seat of renewed activity. We must always be on the lookout for this condition because, with our increased knowledge of tuberculosis, we are coming more and more to the conclusion that infection is a matter of early years and that the first focus in the lung often comes on in early childhood and there remains quiescent until later in life. This degenerative change may be detected by both inspection and palpation of the muscles. It is the sternocleidomastoidei, pectoralis, trapezius, levator anguli scapulæ, and rhomboidei that show it best. The pectoralis often looks smaller than its mate on the healthy side; or, if the disease affects only the upper part of the chest, the line of demarcation between that portion of the pectoralis which is degenerated and that which is not is often clearly visible. When the trapezius is wasted to an extreme degree, the supraspinous fossa appears flatter than on the unaffected side, and that portion which runs up along the cervical vertebræ appears to be wasted and smaller than the same part on the other side. Sometimes, when the degeneration is extreme, the supraspinous portion appears to be decidedly cupped, as previously mentioned. Often, a glance at the trapezius alone from behind is sufficient to make a probable diagnosis of a chronic or quiescent lesion at that apex. Sometimes these degenerated muscles together with the wasting of the subcutaneous tissues cause a very marked change in the appearance of the neck and shoulder. From the wasting the supraclavicular and supraspinous fossæ, as well as the infraclavicular fossa, if the disease is at all extensive, become very prominent and the shoulder, being no longer fully supported, becomes lower than its mate. It is probable that occupations may cause the lowering of some shoulders also, but that a chronic apical inflammation (tuberculosis) will, I have proven to my own satisfaction; for in right-handed individuals, where the chronic lesion is at the left, I find the tissues wasted and the left shoulder lower than the right.

From the rigidity of the muscles in the presence of acute inflammations to the marked degeneration which sometimes occurs when the process has become chronic, there are all grades of change. A common finding on palpation during this transitional stage, which is indicative

of degeneration, is a condition in which the muscle bundles separate easily, when the tips of the fingers are pressed down over them. This is especially common and easily detected in the pectoralis and trapezius. Acute inflammation in an old chronic process is reflected in a special manner by the muscles. The bodies of the muscles are smaller because of the degeneration which has occurred and their power to contract seems to be more feeble than normal; and, as mentioned above, the bundles are somewhat separated. A spasm on their part shows not as a contraction of the same muscles under normal conditions but as a mass much smaller than usual, and sometimes as several distinct masses.

The feeling of resistance felt on palpation of the chest where an infiltration of any marked degree is present, is probably made up of two factors: first, the muscle change; and, second, the increased density of the underlying infiltrated tissues, when compared with the normal. It is often impossible to assign to each of these factors their proper share in causing resistance. Especially is this true when palpating over the flat muscles of the chest, such as the upper portion of the pectoralis, and those covering the lower portion of the chest posteriorly. In such muscles as the sternocleidomastoideus, scaleni, trapezius, and the lower portion of the pectoralis, which stand out more or less prominently so that they can be palpated alone or can be caught up between the thumb and finger, there is little or no difficulty.

The pathology of the degenerative changes which I have described is interesting. It has long been known that the muscles as a whole degenerate as tuberculosis advances to the chronic state, the wasting at times being of an extreme degree. The irritability of the musculature, as evidenced by the contraction of certain bundles when percussed (myoidema), has been looked upon as an evidence of degeneration; and the fact that it occurs in typhoid fever in muscles which are subject to Zenker's degeneration strengthens this view. This view is further strengthened by the fact that it occurs in tuberculosis most frequently after the disease has existed for some time and the muscles have degenerated.

I have examined numerous specimens of neck and chest muscles taken from individuals who were afflicted with pulmonary tuberculosis. While in some instances I had not examined the subjects during life, yet the muscles were the ones which would have shown the change on clinical examination. The pathological examination of these speci-

mens confirm my clinical findings as to degeneration. Quoting from one of my former papers (22), "These muscles show both degenerative and regenerative changes, sometimes the striæ are indistinct, at other times entirely wanting. The muscle fibers take the stain poorly and are at times destroyed. In some cases there is an increase of nuclei, and a tendency to budding, indicating a regenerative process."[1]

There are times when the muscle changes are extremely difficult to determine clinically: for example, when the lesion is extremely small, and not at all or only slightly active; or, when the lesion is more extensive and has become quiescent in a patient with poor musculature; or, when both sides seem equally involved (a very rare condition) in patients with fleshy necks; or, where there is an acute lesion posteriorly and the spasm affects mainly the trapezius and levator anguli scapulæ in a patient who has developed these muscles by extra work beyond that of their fellows on the opposite side. From this it will at once be seen that the difficulties are very few and inconsequential. In all these instances, however, the change in the diaphragm as evidenced by a limiting of the expiratory excursion may be determined and may prove of distinct corroborating value.

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[1] My thanks are due to Professor Ghon and Privat Docent Bartel of the Pathological Institute in Vienna for giving me access to material and making the microscopical examinations for me.

CHAPTER II

Cause of Muscle Spasm and Degeneration

In suggesting a cause for the muscle changes, in my previous papers I found analogy my only guide. The nature of the case is such that a reflex stimulation is the only thing that can be considered. The muscle spasm seems to be analogous to that found in the abdominal lesions, such as appendicitis, gastric ulcer, cholelithiasis, and peritonitis, which is usually described as Nature's method of protecting the injured part.

After examining carefully into the extent of the muscle change, it is evident in the case of the chest muscles, the same as in that of the abdomen, that the spasm and degeneration which occur do not follow any particular nerve distribution. Where an entire muscle or several muscles are supplied by one nerve, only a part of the muscle or one of the group may be in spasm or show degeneration, while the other part or other muscles of the group may show no change. The irritation seems to follow certain fibers of the nerve rather than the nerve as a whole. Thus, in appendicitis the muscles of the lower right quadrant of the abdomen are in spasm while others supplied by the same nerves are not; and, in acute pancreatitis, a portion of the left rectus is thrown into spasm, while other portions of the muscle supplied by the same nerve are not. The same is true in angina as pointed out by Mackenzie. Certain portions of the precordial region are thrown into spasm while other parts of the same muscle, supplied by the same nerves, are not.

The same thing seems to hold in the neck and chest muscles, and is especially demonstrated in the pectoralis in old chronic lesions where the muscles have undergone degeneration. Not infrequently the upper portion of the muscle will be degenerated to such an extent that it not only can be detected by palpation but is even plainly visible to the eye; and yet the lower portion will apparently be in normal condition.

The degeneration of the muscles in clinical tuberculosis has long been known, but most observers have recognized it only as a general

condition, hence considered it as having a constitutional cause. The cause usually assigned has been the general wasting which occurs as the disease advances or the toxemia resulting from the tuberculous infection. That these play their part in the tissue wasting in tuberculosis can not be denied, but they do not account for the localized degenerations which I am describing. Nothing but a local cause will satisfactorily account for these; and this we find in the irritation of the cells in the segments of the cord from which the nerve fibers supplying the muscles take their origin, as a result of the stimulation coming through the sympathetics from the inflamed lung. Whether the degeneration is due to reflex overstimulation of the motor nerves, as mentioned elsewhere, or a reflex trophic disturbance, I am not entirely sure—probably both are factors. That a trophic disturbance is a part is indicated by the fact that the subcutaneous tissue also wastes, although it is not endowed with motor power.

It is worth noting in passing that the muscles which seem to show the greatest amount of spasm, the trapezius and sternocleidomastoideus, are both supplied by the spinal accessory nerve. This fact and the fact that a branch of the spinal accessory is also found in the vagus suggests another path besides the sympathetic.

As important evidence bearing on the distribution of the muscle change I would cite Sherrington's work as recorded by Mackenzie (29): "Professor Sherrington has recently been making an investigation into the effects produced in the abdominal muscles by stimulating the sympathetic nerves. He dissects out and divides a branch of the solar plexus going to the bowel, and stimulates the central end. There is an immediate response in the broad muscles of the abdomen, which contract over an extensive area. By dividing one after another the posterior roots of those spinal nerves that supply this extensive area, the extent of the contraction becomes greatly limited until when there is but one posterior root left intact, the contraction becomes limited to a few fibers of the muscle."

It has been suggested that the muscle change might be due to a direct stimulation from the pleura; and I am surprised to find that a great many physicians when asked for an explanation for the muscle rigidity in inflammations of the abdominal organs, give as the cause a localized inflammation of the underlying parietal peritoneum. That this is not the true explanation is suggested by the fact that these

muscle spasms occur in appendicitis, pancreatitis, and inflammation of the various viscera when the disease is confined to the organ itself and when the organ is far removed from the anterior parietal peritoneum. On this point I wish to quote from Piersol (27): "It is of interest and importance to observe that those intercostal nerves corresponding in their origin from the spinal cord with the ganglia giving off the splanchnics, together with the first two lumbar nerves, the ilio hypogastric and ilio inguinal, supply the abdominal wall with motor and sensory branches. In this way the same segments of the spinal cord supply the abdominal viscera as well as the skin and muscles over them. A similar arrangement of the nerves is seen in the joints, where the same nerves supply the skin covering the joint, the muscles which move it and the joint structures. As a result of this, when necessary, all parts of the joint act in sympathy."

The spasm and degeneration of the muscles of the neck and chest, which I have described, are unquestionably of different origin from the muscle spasms found in tuberculosis of the joints and the degeneration of the muscles in cases of tuberculosis of the bones as described by Neihans (28). In cases of the joint, at least, the irritation is probably direct, for, as Piersol states, the joint and the muscle and the skin over it are supplied by the same nerve. Niehans, however, is of the impression that in the case of tuberculous foci in the bones the muscle atrophy is of toxic origin, as will be shown from the following quotation from his paper: "For at least a decade we have been interested in attempting to make clear the relationship existing between the atrophy of muscles and the tuberculous focus in the underlying bone and to see if this condition follows any certain law which can be made of diagnostic importance. My observation would indicate such a possibility: *at times the focus in the bone corresponds closely with the point of insertion of an individual muscle or a group of muscles which show atrophy.* An example might serve to illustrate the point—a patient fifteen years old suffering from tuberculosis of the skull bones. He showed at the same time evident atrophy of the biceps femoris with thickening of the tendon sheath, especially of the short head where it is inserted into the head of the fibula. (A tuberculosis focus was found in the latter.) That this disease was not the cause of the atrophy as is so often believed was shown by the fact that the patient ran upstairs two steps at a time. Since, in such cases, we have never been able to find a change in the normal dark brown color of the muscle substance,

it seems that we are warranted in considering this atrophy as due to toxic action."

Jessen (13) recognizes the presence of atrophy of the trapezius in tuberculosis and considers it probably due to neuritis. He does not mention the spasm of the muscles, however. I quote from him:

"The question must also be considered to what extent those particular muscle atrophies of the trapezius, where either one or both apices are involved, are the expression of a toxemia or are the result of an underlying neuritis.

"The same question must also be considered with reference to thorax paralyticus, for it seems certain that this does not furnish the conditions for a tuberculous infection but is rather the sign of a tuberculous infection being already present, oftentimes even from early childhood.

"This belief has been expressed by Cohnheim. While no anatomical investigation of this subject has been made, yet the following considerations must be taken into account. If this muscle atrophy were only the expression of a general intoxication, it is impossible to see why other muscles in this early stage should not also show atrophy; but, we have seen that, in beginning apical tuberculosis, the atrophy always appears to affect only the trapezius muscle, so that oftentimes, even in cases which are not the seat of contraction due to advanced tuberculosis, a glance will show the seat of the disease.

"There must, therefore, be a regional relationship between muscle atrophy and pulmonary tuberculosis. If belief in the diffusion of toxins in the diseased areas was to be considered, it would have to be looked upon as being hypothetical; but, we know, as a matter of fact, first, that a latent neuritis is often present; second, from the investigations of Head and Egger, as well as from many observations made by myself, that it is known that a certain relationship exists between pulmonary tuberculosis and the nerves of the thorax, leading to a clinically demonstrable disturbance of the sensation of the skin. It does not, therefore, seem improbable that the nerves, which regulate nutrition and muscle tone, should be affected in this same manner; and, that this circumscribed muscle atrophy could also be produced through irritation of the nerve whether it be of the nature of a latent neuritis or a purely functional disturbance."

Head (7) described conditions of the sensory nerves of the chest analogous to those of the abdomen, showing how inflammations of the internal viscera cause disturbances in sensation and trophic changes

in certain areas of the skin. These areas do not follow the distribution of any particular nerve. In this discussion I think it is well also to mention the work of Sherrington, for his observations were carried on differently and his results are also different. Sherrington traced out the distribution of the posterior dorsal roots of the cord by cutting all the roots except the one he was investigating. He then tested the skin to touch and in this way determined the area supplied by each posterior root. As a result of his investigation, he found that the roots overlap and that the same area of skin is supplied by more than one root.

Head's investigations were different. He tested the various areas of the skin with reference to pain, heat, cold, and trophic changes, and found that these areas did not overlap to any extent. To account for the difference in these two results, Head studied the areas of eruption in herpes and the sensory changes in conditions where certain segments of the spinal cord were injured and compared them with those where the roots were injured. He came to the conclusion that between the two there was a fundamental difference. Sherrington's areas represent the cutaneous distribution of the posterior spinal roots and overlap, while Head's areas, shown in Figs. 5 and 6, represent areas supplied by segments of the cord and do not overlap to any extent.

Study of cases of herpes and spinal cord injury convinced Head that the areas of eruption in the former and the areas of analgesia in the latter bear close relationship with the areas of tenderness in visceral inflammations. In the sensory areas, described by Head (Head's zones), in visceral inflammations the tender zones represent areas supplied by segments of the spinal cord rather than nerve roots.

"For we must suppose," says he, "that a sensory disturbance passes up the nerve fiber from the viscera to set up changes in the activity of that segment of the spinal cord in which they terminate; and that these disturbances of activity are referred to that portion of the skin which is supplied by the affected segment. And when we turn our attention to the results produced by localized organic lesions of the spinal cord, we find that the affected areas closely correspond to those which appear in functional disturbances of the viscera."

It would seem but natural that this same segmental stimulation of the cord would affect the motor nerves as well as the sensory and this explanation comes nearer accounting for those muscle spasms in dis-

eases of the abdominal viscera, and the one described by Mackenzie in angina, as well as the ones which I have described in diseases of the lungs and pleura, than any other that has been advanced.

Owing to the visceral stimulation we are warranted in assuming that

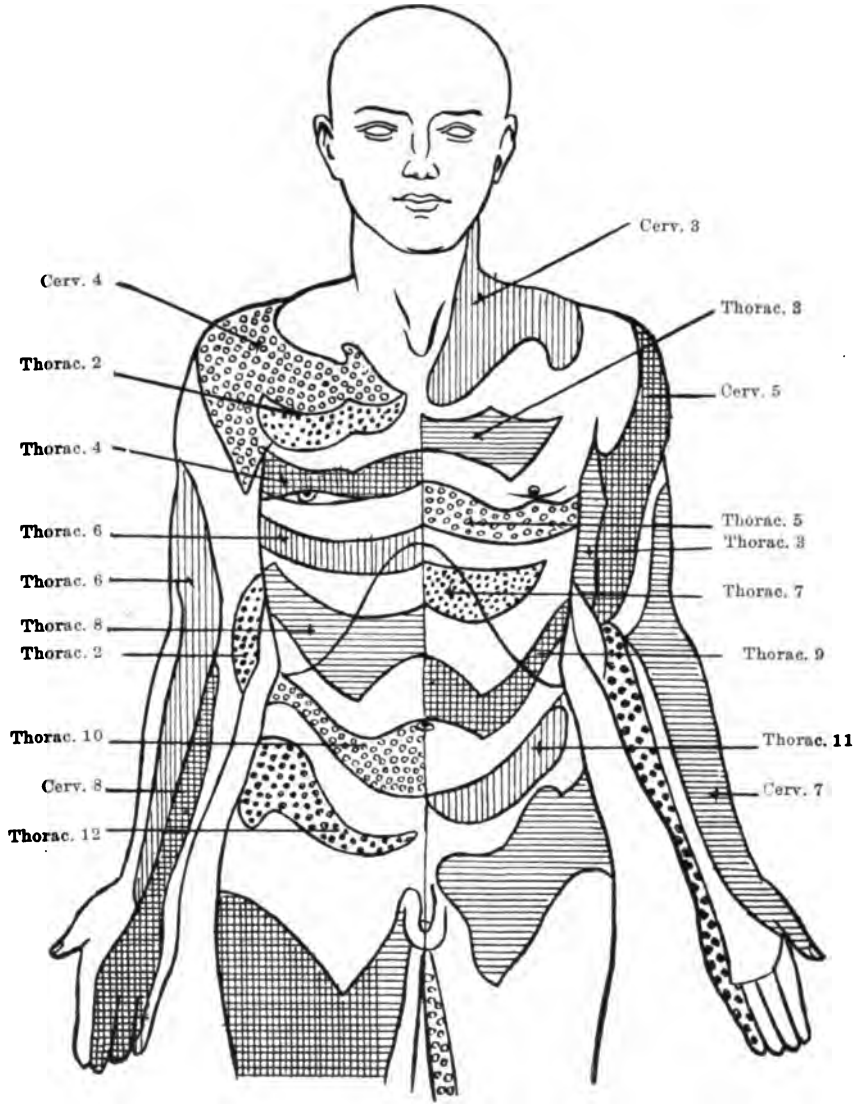

Fig. 5.—Head's Zones. (Anterior surface of the chest and abdomen.)

the segment of the cord is in a state of heightened activity, as is shown by the reflex sensory skin disturbances so carefully worked out by Head, and it would not be at all unnatural to suppose that the stimulation should also affect the motor and trophic fibers. Mackenzie (29)

accepts this as the explanation of the abdominal and heart reflexes; and, while I have not made the careful experimental studies that would definitely warrant a positive assertion, yet I consider that I am justi-

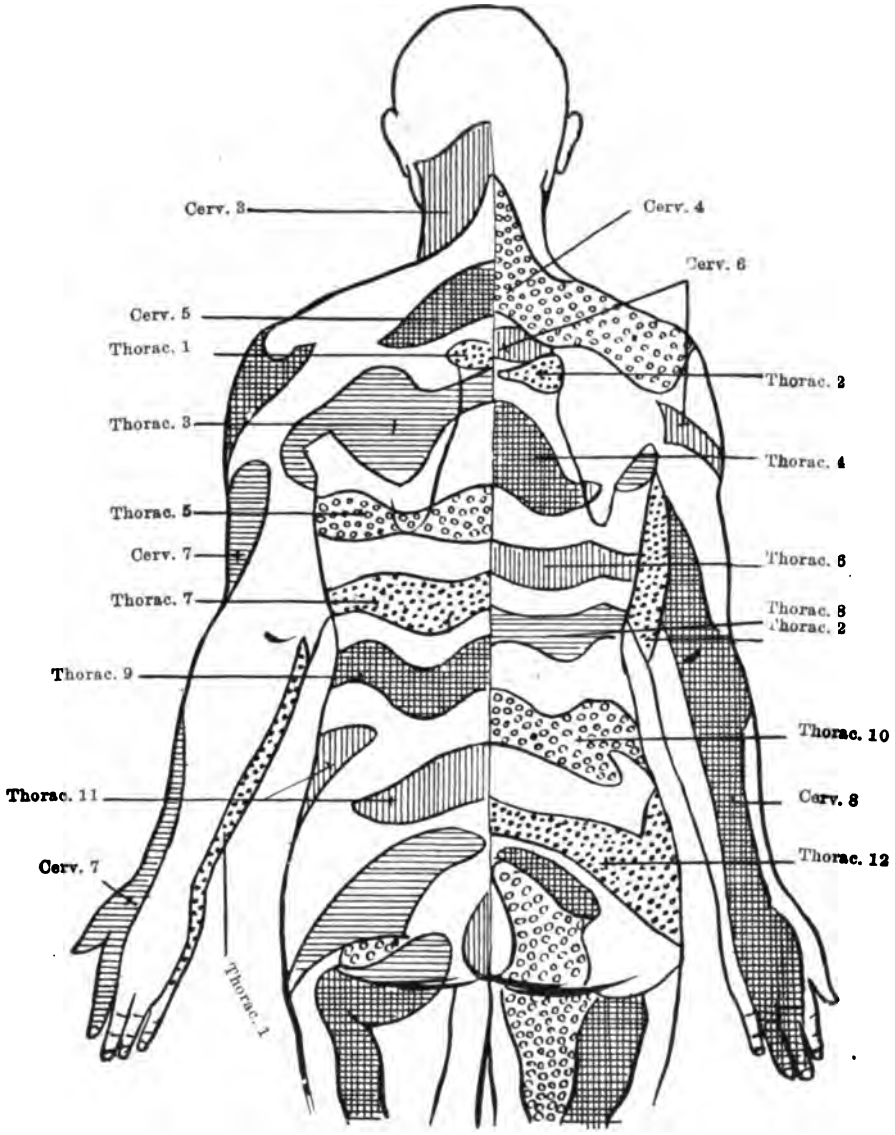

Fig. 6.—Head's Zones. (Posterior surface of the body.)

fied in assuming that the spasms and degenerations affecting the muscles of the neck and chest are of the same segmental origin. Fig. 8 (p. 51) shows the path of the reflex stimulation from the viscera to the muscle and skin through the cord.

The cause of muscle degeneration I have discussed in my previous papers. It would seem that the degeneration might result from the muscle being held in spasm for a prolonged time. It is contrary to physiological law that a muscle should remain indefinitely in a state of contraction and preserve its normal texture. Such a contraction would necessarily have degenerative change as its consequence. I look upon these muscle degenerations as being due at least partly to the continuous irritation caused by the impulse from the lung and pleura, keeping up the muscle stimulation over a long period of time, and, as being similar to the degeneration following excessive work, although I think we have reason for believing that it might be partly a trophic disturbance analogous to the motor and sensory changes.

Adami (30) in speaking of overwork says, "If the work of the cell is excessive, exhaustion of the cell will set in, the cell substance will be broken down more rapidly than it is formed, and the cell as a whole will shrink." And again, "A similar atrophy (similar atrophy from disuse) may result from excessive function. Here again the best examples occur in connection with the neuromuscular system, special groups of muscles and their nerve centers, which are overworked in the performance of certain movements, are apt to exhibit, following upon the hypertrophy which has resulted from increased work, the onset more or less rapidly of atrophy. Neurons, or it may be the muscle fibers, become exhausted and worn out, and as a consequence we have paralysis with atrophy of the involved muscle." An example of this is the atrophy of the muscles of the blacksmith's arm. The fact that in such a disease as tuberculosis the entire musculature of the body is suffering from the effects of toxins might make these muscles which are in a state of chronic spasm, more prone to degeneration; but, the toxin theory cannot account for the fact that the degenerative change is so selective in its action as to affect only or even principally those muscles or parts of muscles which were previously in spasm.

I realize that there is a large subjective element in every physical examination. Unfortunately, this affords opportunity for differences of opinion and error. It has seemed to me in my physical examination that the muscle spasm, likewise the later muscle degeneration, follows quite accurately the areas of involvement; yet this may be only partially true, for it is a very difficult problem, in fact impossible, to always be able to accurately determine by the most careful examination the extent of the finest changes in a pulmonary lesion. Furthermore,

it is almost impossible when palpating over the flat muscles of the chest to differentiate the increased resistance noted on account of the increased tension of the muscle and that produced by increased density of the tissues caused by the infiltration of the lung tissues. Whether the muscle change is coextensive with the area of involvement or not, those who have worked with me have been convinced that the change on palpation affords information as to the extent of the lesion which is at least as accurate as that obtained by our other methods employed in physical examination.

The following cases will illustrate the harmony between the muscle change and the findings on percussion and auscultation.

Case I: Female, age 24.—Had always been well until three months previous to examination, when she began to feel tired, showed a capricious appetite with digestive disturbances and the loss of four or five pounds in weight. She had a slight hacking cough and temperature of 99.4. She consulted a physician who prescribed a tonic, thinking it was only a slight indisposition. After showing no particular improvement she requested that an examination of the chest be made. Nothing was found. There was no sputum, but a Von Pirquet skin test showed a prompt reaction. It was now that she sought my advice. On inspection I noticed that the muscles of the anterior portion of the neck and chest were symmetrical; posteriorly the left trapezius and levator anguli scapulæ were more prominent than the same muscles on the right side. Palpation confirmed this and revealed the fact that these muscles were very resistant to the touch and gave evidence of being in distinct spasm. Inquiry showed that this could not be due to occupational influences. Probable diagnosis from muscle change and clinical history: a slightly active tuberculous lesion at left apex.

Percussion revealed an increased resistance and impaired note due, at least in part, to the altered muscle. Auscultation showed a slightly roughened, harsh, and weakened inspiratory note over the posterior aspect of the apex corresponding to the muscle change. Diagnosis: Incipient active tuberculosis of the left apex, confirming the probable diagnosis made from the condition of the muscles.

Case II: Male, age 28.—Had always been well until two weeks previous to consulting me, when he had spit up several mouthfuls of blood on three or four successive days. Previous to this he had neither coughed nor had he shown any other symptoms indicative of tuberculosis. Three physicians had examined him and given their opinion

that his lungs were perfectly free from disease. Physical examination revealed on inspection slight lagging of the left apex with a deepening of the supraclavicular notch and an undue prominence of the clavicle, the tissues about the apex being somewhat wasted. The clavicular portion of the sternocleidomastoideus and the scaleni of the same side stood out like cords. Palpation showed these muscles to be distinctly rigid and in spasm. Probable diagnosis from the condition of the muscles and clinical history: active tuberculous lesion at left apex in the seat of an old focus. Percussion showed increased resistance to the finger and slightly impaired resonance. Auscultation revealed slightly roughened inspiration with prolonged expiration and a few fine rales on coughing. Von Pirquet skin test was positive in twenty-four hours. Diagnosis:—New activity in seat of old lesion, at left apex.

Case III: Female, age 20.—Three years previous to present examination had been sent out to the desert because of a tuberculous lesion in right apex from which she had apparently recovered. Patient remained well until four months ago when she began to feel tired and depressed; lost four pounds in weight, had a slight hacking cough with some expectoration of whitish mucus in the morning. Examination revealed:—Inspection: lessened motion at both apices, more marked at left. Head slightly bent to the right. Right supraclavicular notch depressed and tissues wasted, the muscles not prominent. Right pectoralis looked degenerated in upper portion. Left supraclavicular notch full, almost even with the clavicle; sternocleidomastoideus, both sternal and clavicular portions, and scaleni prominent. Posteriorly on the left the trapezius, levator anguli scapulæ, and rhomboidei, prominent; muscles on the right apparently normal. Palpation: neck and upper chest muscles on right showed loss of normal elasticity and the pectoralis felt thinner than normal. They were less prominent than the muscles of the left which showed increased resistance. Probable diagnosis from condition of muscles and clinical history: old chronic or healed lesion at right apex with new acute process at left apex. Percussion showed increased resistance and higher pitch to second rib anteriorly and spine of scapula posteriorly on the right and to second rib anteriorly and the middle of scapula posteriorly on the left. Auscultation: right apex anteriorly down to second interspace showed harsh inspiration with harsh prolonged expiration, the signs indicating an old healed lesion. Posteriorly, apparently normal. Left apex,

anteriorly, to second rib and posteriorly above spine of scapula and in the upper portion of the inter-scapular space, showed slightly rough harsh inspiration, with prolonged expiration, the note being slightly weakened. Diagnosis:—Old healed lesion at the right apex, with new active lesion at left apex, occupying the upper part of the upper lobe and the apex of the lower lobe.

These cases could be multiplied many times from my records, but they are sufficient to show that this muscle spasm is of value in diagnosis and especially in early diagnosis; and, second, that it seems to be accurate in its distribution over the parts involved. I will illustrate the value of the muscle degeneration as a sign of disease more fully when I discuss the advanced lesion in the lung.

Regarding the muscle changes in the neck, it is possible that there may be other organs which, reflecting into the neck, cause muscle spasm; but, that the lungs do, there can be no doubt. From our analogy of Head's sensory zones in the neck, we must assume that there are probably others. I will quote again from Head (7), who refers to the experiments of others, as well as his own: "Edgeworth finds that the heart, lungs, liver, stomach, and intestines are all supplied by large fibers from above, which reach them in the vagus, and in the case of the heart, by the depressor nerves. Now these are the organs which I believe give reference into the areas above the clavicle, i. e., into the region of the head and neck. . . . The most definite reference into the neck is given by the liver, lungs, and heart."

If the motor nerves reflect in the same way as the sensory, then we could have spasm of the neck muscles from diseases of the heart, liver, stomach, and intestines; but my practical experience, which has been almost entirely with tuberculous cases, but which has also afforded me many other complications on the part of other organs, has taught me to place great reliance in the neck muscles as diagnostic signs in inflammation of the apices of the lungs. Even if the other organs do reflect in the neck, it does not vitiate the muscle sign. It only requires differential acumen.

It would be worthy of study to learn what effect, if any, enlarged and inflamed mediastinal glands, especially in childhood, might have upon the systemic muscles and especially upon the production of curvature of the spine; for, in the light of our new studies, we are forced to consider reflex phenomena as important causes of bony deformities.

In the discussions which have resulted from my publications on this

subject, there have been a variety of suggestions as to the cause. I must admit that I have been at a loss to find an accurate explanation which had been already worked out. In the first place, as far as I am able to learn, physiologists have not yet described this motor reflex for the lungs; consequently I have been compelled to grope somewhat in the dark. But now it seems to me that the explanation must be similar to the one which Head gives for the sensory reflexes on the skin and which he has well worked out for the chest and neck.

Hart (31) in replying to the argument in one of my former papers (22) takes issue with my suggestion that the muscle spasm is a reflex stimulation passing from the inflamed lung and pleura to the cord and then back through the motor nerve to the muscle, and thinks that the condition of the muscles which I am describing is due to a hypertrophy from overwork as described by Freund and his followers (1, 2, 3, 4, 5), and suggests that the muscle change might be due to toxic action but questions the likelihood of a toxic effect being so selective in its action. I replied to Hart's argument in a subsequent paper (32) and shall discuss his theory more fully when I refer to the ankylosis of the costosternal joint, and shortening of the first rib as a predisposing cause of tuberculosis.

Bönniger (15) exhibited patients before the Berlin Medical Society on May 11, 1910, demonstrating a condition which he calls unilateral lymph stasis and described it as a condition affecting the skin, subcutaneous tissues, and muscles in such a way that the tissues appear to be increased in volume and to be firmer than normal. He gave as the explanation of the phenomenon, a lymph stasis due to a diseased pleura. He considers this condition to be the same as that which I have described as muscle spasm, rejects my explanation of a "reflex causation" and suggests his explanation as being more probable. His explanation, however, in no manner explains the phenomena which I am describing.

Orszag (33) confirms my clinical findings on the part of the muscles and refers to a very interesting study made by Bálint (14) upon the electrical reaction of the muscles in the presence of pulmonary tuberculosis; quoting from Orszag's paper, Bálint found as follows: "That the musculature of patients was, in the greater number of cases, highly irritable. Sometimes weak faradic currents which were not felt would produce contraction. The increased irritability of the muscles was on that side of the body where the disease was located. In cases where

both sides were affected, the greatest irritability was found on the side of the severest disease. The irritability of the muscles decreased as patients improved as determined by the physical examination. Patients in the second stage showed the greatest irritability, the third and first showing less."

Bálint's explanation is as follows: "The impulse from the lungs passes into the cord through the posterior horn, and thence to the periphery, causing the disturbance noted. A portion of the irritation is apparently carried through collateral fibers to the anterior horn of the same side so that its cells are in a state of constant irritation which shows in the reflex contractions of the muscles. On this account these muscles become so sensitive that they react to a much weaker irritation (the electrical stream) than the muscles on the side of the body free from disease, where centers are influenced to a lesser degree or not at all. The highest grade of increased irritability is to be found in those muscles which receive their motor fibers from the same portion of the cord which supplies nerves to the apices of the lungs. The participation of the other muscles depends upon the manner in which the irritability happens to be radiated."

This contribution of Bálint's is very important in that it demonstrates that the motor nerves are influenced reflexly in diseases of the lungs and furnishes additional proof to those who doubt the presence of muscle spasm and degeneration as clinical signs of reflex origin from the lungs. He also accounts for the condition in a similar manner to that which I have suggested. What Bálint requires electrical apparatus to determine can also be determined by inspection and palpation, as shown by me.

Orszag in the paper quoted states: "Upon the basis of my examinations which I made in part on ambulant cases, and in part in the tuberculosis wards of the clinic, I can confirm the findings of Pottenger *in toto*. For the most part the rigidity of the musculature on the side of the tuberculous disease of the apex was very pronounced."

Orszag's conclusion "that rigidity of the muscles in pulmonary tuberculosis is confirmed but is of no special value as an early diagnostic sign," seems strange after the above statement, and shows that, while he appreciated the muscle change, yet he has not grasped it fully; for, if there is one place where I have found this muscle spasm to be of value it is in early diagnosis of apical tuberculosis; and, as illustrated by the cases mentioned above, it is present before any marked symp-

toms appear in the lung. He thinks Bálint's determination of the electrical reaction of the muscle is preferable as a method of determining the activity of the disease. It is probable that the relative importance of my sign may grow in his estimation with greater familiarity in its determination.

In support of my view that muscle spasm is an important sign in early active apical tuberculosis and that the condition of the muscles gives very important differential diagnostic evidence between active and inactive lesions I would cite the work of so careful and experienced an observer as Wolff-Eisner (34) who has made a very careful study of early diagnosis and the differentiating of active from quiescent lesions. He compared the muscle findings with the conjunctival tuberculin reaction in his large hospital material and concludes that the great practical worth of light touch palpation in the recognition of muscle change is "in the recognition of initial active tuberculous processes" and further says that the muscle change in combination with the conjunctival reaction offers a great aid in the solution of that question which is of so much meaning for the therapy of tuberculosis: viz, the determination of the presence of active tuberculous processes.

Bredow (35) examined 107 patients suffering from pulmonary tuberculosis, 78 of whom were in stage I, 23 in stage II, and 6 in stage III. He endeavored to determine the presence of the muscle change as I had described it in my various papers. While he found it in some instances yet he arrived at the conclusion that it is of no particular value in early diagnosis. As far as cause is concerned he rather favors the opinion that it is partly due to irritation from pain and that it represents an effort of nature to put the part at rest to avoid pain; and that it is also partly a result of anomalies in the upper aperture of the thorax.

In reference to the muscle spasm being an attempt of nature to put the part at rest in order to avoid pain, it is necessary to note that in incipient tuberculosis the process is largely free from pain. Regarding the relationship of muscle change to the anomalies of the upper aperture of the thorax, I will describe these later, and will refer my readers to that portion of my paper.

Cyriax (36) confirms my findings and in a very interesting paper calls attention to his own observations which are entirely in harmony with those of my own. He also mentions that he had described this condition as early as 1903 (12). On page 163 he says: "In the ab-

domen reflex contraction of the muscles of its wall are frequently found as a result of irritative or inflamed conditions of organs in its cavity. The same state of matters obtains with the thorax; reflex contractions of the intercostal muscles result from many morbid conditions of the lungs, bronchi, and pleura." On page 312 he says: ". . . . the impediments to respiration that lie in the muscular apparatus. These are found to be: contractions of the intercostal muscles over the affected lobe or lobes, deficient action of the diaphragm, with or without marked contraction of the abdominal muscles." On page 314 he mentions that in a case of lobar pneumonia affecting the left lower lobe there was a contraction of the fifth to ninth intercostal spaces. He further states on page 313: "In two cases of pneumonia that I have treated, the kidney region of the affected side was much more tender to touch than that of the opposite side."

I am glad to give credit to this independent observer at this time, although I was not aware of his work until after my early publications. Cyriax, however, did not recognize the condition as of diagnostic importance, but spoke of it as being a condition requiring therapeutic attention; and while I have not seen his original article, I am led to believe that he at first only noted the change in the intercostals. It is strange that I also noted it first in the intercostals, as is shown by my first two papers; but, after thoroughly appreciating the change, I recognize it now in the superficial muscles as being much easier of detection than in the intercostals. Thus, it is surprising that both he and I found the spasm in the muscles where it is most difficult to detect, before we found it in the more superficial muscles where it is easier of detection.

That some of the observers quoted above, who have attempted to detect the muscle change, have been unable to satisfy themselves as to its value, is not surprising. It is interesting to note, however, that nearly all have been able to find it in some instances. Wolff-Eisner, Orszag, and Cyriax have been able to confirm it *in toto*. My experience has been, since the publication of my first paper, that those who have seen my personal demonstrations have been able to detect the spasm and degeneration in their own work, while many who have attempted to work it out alone have failed. A sufficiently large number of workers, however, have been able to detect the change without demonstration to establish the practicability of the sign beyond doubt.

One of the difficulties in assigning to this muscle change its true

position as a diagnostic sign is the fact that we ask examiners, who have been in the habit of inspecting and palpating chests without deriving much information from the procedure, to look at and palpate the same chests and note a condition which is so evident after it has once been comprehended that it offers a valuable sign for the determination of intrathoracic conditions. When the examiner looks at the chests again, being prejudiced by his training and experience, the muscles look and feel the same as they did before, consequently the change is not fully grasped. But, if students are taught that the muscles of the neck and thorax reflect the inflammations of the intrathoracic organs, in the same manner as they have been taught that the abdominal muscles reflect the intra-abdominal inflammations, then it would be learned as a part of their education and be fixed as a part of their diagnostic routine. After an extended experience in the employment of the muscle change in diagnosis of disease of the lungs and pleura, which now extends over more than three years, during which time I have employed it in making several thousand careful examinations, I feel justified in saying that there is no other physical sign that is so easy for me to determine and no other single sign that suggests so much as to the condition of the intrathoracic organs as does the muscle change. I believe when fully recognized it will offer more suggestive information to the practitioner, who is not accustomed to making constant chest examinations, than any other sign.

CHAPTER III

What Can be Determined by Noting the Condition of the Neck and Chest Muscles in Acute and Chronic Inflammations of the Lungs and Pleura

From the three case histories detailed above illustrating the manner in which the muscle change seems to follow that of the intrapulmonary lesions, it will be seen that in these early cases the changes are principally above the clavicle and angle of the scapulæ, and that by noting the condition of the muscles over this area the examiner can form a fairly accurate idea of the presence or absence of disease. Tuberculosis usually begins at the apex or near the apex of the lung; and, it has been my experience that the muscle change in such cases is confined to the neck muscles, the upper intercostals, and the superficial muscles or portion of muscles covering them, depending upon the extent of the lesion. The only change that we expect to find in a primary active lesion is muscle spasm. If the lesion represents activity in an old quiescent lesion, however, then we must expect both degeneration and spasm.

When a patient presents himself for examination, he should be stripped to the waist and asked to seat himself comfortably with hands to the side, his head looking forward in his usual natural manner, relaxing all muscles. This relieves all undue and unnatural tension that might be thrown on any muscle or group of muscles. Any lack of symmetry about the neck muscles, either anteriorly or posteriorly, should call attention to probable trouble in one or both lungs. If we will recall what we have written above, we will see that if the patient is suffering from an active tuberculous lesion at one apex in a chest previously free from disease, the sternocleidomastoideus and scaleni or trapezius, singly, or in combination, will usually stand out more prominently and be more resistant to the touch than the same muscles on the other side, the degree depending upon the acuteness and the extent of the lesion. When touched lightly it will be seen that their fibers are on an increased tension. Muscular spasm is the motor expression of the inflamed lung and we accept it as being

produced by the inflammation in the lungs sending impulses through the sympathetic nerve fibers to the cord where they impart to the cells of the segment on the side of the involvement a certain amount of irritability, which shows itself peripherally, through the posterior horn, in changes in sensation as described by Head, and, through the anterior horn, as muscular spasm and degeneration as here described. An increased rigidity of these muscles must then be taken as a reflex expression which demands that the seat of the primary irritation causing the stimulus be found. In these early cases that I have examined; with one or two exceptions, I have found the seat of the stimulus in the apex of the lung on the side of the muscle change. We must bear in mind, however, that there is a possibility of the muscles on the opposite side from the lesion being thrown into spasm as mentioned above; but my experience indicates that this occurs extremely rarely. If the apex, which is now the seat of involvement, was previously the seat of a quiescent lesion, then, as a rule, the supraspinous fossa is depressed as a result of contraction and tissue degeneration; the muscles under these circumstances are somewhat degenerated and naturally the spasm is not as marked as in a primary involvement. The trapezius will usually show, instead of one large rigid mass, a mass smaller than normal composed of several distinct rigid bundles of the muscle between which the tips of the fingers can be inserted. This condition I have learned to recognize as being caused by the process of degeneration and to be evidence of a past or a chronic inflammation of the underlying lung tissue.

Another sign usually noted in cases where there has been an old lesion is a bending of the head to the side. The bending evidently takes place for the most part between the skull and the first cervical vertebra, although the cervical vertebræ as a whole are sometimes bent to the side. This is probably due to the action of the rectus lateralis, as it is shortened by degeneration in the former instances, and to a combination of many of the neck muscles when the entire cervical spine is turned. This is readily understood, for, in as much as all of the neck muscles receive nerve supply from the cervical segments of the cord, the same segments which give off the sympathetic branches to the lung, all the neck muscles may be involved in spasm as a result of the reflex stimulation from the inflamed lung. This bending of the head is often very pronounced in advanced cases. The head seems, as far as I have been able to determine, to nearly al-

ways bend to the side of the severest infection or the oldest infection, provided it is of considerable magnitude. Sometimes the head is not only bent to the side, but the chin is turned to the opposite side, owing to the shortening of the sternocleidomastoideus. Occasionally when both lungs are involved we see the entire head pulled down and forward by the action of both sternocleidomastoidei acting conjointly.

The existence of what is known as round shoulders must be considered in connection with these muscle changes. A great deal of useless energy has been spent in trying to prevent the tuberculous from becoming round-shouldered. They have been implored to straighten up; they have been compelled to wear shoulder braces; they have been ordered to spend a certain amount of time with head erect, spine straight, carrying weights upon the head. All of this has been of little avail. The head and shoulders have continued to drop forward. Changes, such as these, must be due to the exertion of some constant force of considerable power. Since it is the muscles that hold the various parts of the body in position, it is to them that we naturally turn for the explanation of this phenomenon. Heretofore it has been thought that the chief factor in its production was a weakening or degeneration of the posterior muscles which hold the spine and head erect, but judging from the cases which I have been able to study, I am inclined to believe that shortening of the anterior muscles through spasm and degeneration, together with lessened mobility of the thorax, are also important factors. Looked at from this standpoint (omitting some cases probably caused by occupational influences) round shoulders must be taken as an expression of a pathological condition of some of the intrathoracic viscera.

To illustrate what pathological conditions muscle changes reflect in more advanced conditions, I will cite the following cases:—

Male, age 28.—Never very strong. Had worked under mental strain with irregular hours for years. During the past two years has had colds more or less frequently, and has been somewhat run down; yet showed no active symptoms on the part of the lungs calling for examination, until four months before consulting me, when he was taken with what was considered a bronchitis. This condition did not yield to ordinary measures; patient lost weight and showed increase of expectoration which upon examination revealed tubercle bacilli. Temperature never above 100.

Physical examination showed the following:—Inspection: head bent

to right; right supraclavicular and infraclavicular fossæ depressed, the muscles appearing to be degenerated; subcutaneous tissue wasted; trapezius in supraspinous fossæ wasted, the shoulder appearing somewhat flatter than normal. The muscles of the neck on the left showed very plainly; the clavicular portion of the sternocleidomastoideus and scaleni being quite prominent and cord like; trapezius, levator anguli scapulæ and rhomboidei apparently fuller and more prominent than normal. On palpation the neck muscles over the right apex and that portion of the pectoral over the first intercostal space felt doughy and degenerated. The anterior neck muscles on the left side were rigid and tense. The posterior group on the left which appeared full on inspection were also more rigid than normal. The entire pectoralis on the left was tense and felt the same as when it is voluntarily contracted. The axillary edge was sharp to the fingers. The intercostals were resistant and board-like. From this the following probable diagnosis was made: an old chronic or healed lesion at the upper portion of the right lung, with an acute progressive process in the upper lobe of the left lung, occupying the entire upper lobe and probably an acute inflammation of the pleura. Percussion showed impaired resonance and increased resistance to the finger over the right lung to the second interspace and over the left lung from the apex to the fifth rib anteriorly. Auscultation revealed harsh inspiration and prolonged expiration over the part of the right lung showing percussion change and a decidedly rough, harsh inspiration, with prolonged expiration and many fine rales on the left side to the fifth rib anteriorly, and down to the upper third of the scapula posteriorly. Aside from the lung condition there was on the left an acute inflammation of the pleura with pleural rub in the region of the third and fourth ribs anteriorly. The diagnosis made by percussion and auscultation fully confirmed the one made by the examination of the muscles. While these latter methods added more accurate information and gave a more detailed idea of the condition of the thorax than inspection and palpation, yet we were able by the muscles alone to determine that the right lung had been previously affected, that the condition was inactive and that the lesion in the left lung was a new one and active and that acute pleurisy was probably present.

To show the muscular findings in a far advanced case I will cite the following:—

Male, age 30.—Was examined by me three years ago, at which time

he had a tuberculous infiltration at the right apex extending to the second rib. Patient rested for a time and then continued living under favorable circumstances and felt fairly well until about a year ago, when he began to feel badly owing to the extension of the disease to the other lung.

Examination at present shows:—Inspection: head bent to right, right clavicle prominent, supraclavicular notch deepened and infraclavicular space flattened; subcutaneous tissue wasted. Right pectoralis wasted to third rib, apparently normal below. Left sternocleidomastoideus and scaleni prominent, pectoralis wasted throughout. Posteriorly, trapezius and levator anguli scapulæ wasted on both sides, more marked on the right where the supraspinous fossa shows as a distinct depression. Palpation confirmed the wasting of the upper portion of the pectoral on the right and the apparent normal condition of the lower portion of the same muscle, the upper portion being thin and doughy (without elasticity) while the lower portion seemed to retain its elasticity. The left sternocleidomastoideus and scaleni were wasted but feebly resistant and cord-like, while the entire left pectoralis was thin and doughy. The trapezius and levator anguli scapulæ on both sides were thinner than normal. Their bundles were easily separated and they felt doughy to the touch. Besides this, the fleshy portions of these muscles were somewhat firmer than normal on the left. Probable diagnosis from muscle condition: an old chronic or quiescent focus on the right to the third rib and a chronic process occupying the upper lobe on the left, which was the seat of activity.

Percussion showed altered note and increased resistance, coextensive with the muscle change. Auscultation showed fibrosis with a few fine rales to third rib anteriorly and to spine of scapula posteriorly on the right, and a rough harsh inspiratory note, accompanied by medium and fine rales with prolonged expiration over the entire upper lobe on the left side. There were localized areas of softening here and there. Patient was losing in weight and strength and having daily temperature of 99.6 to 100. Diagnosis from auscultation, percussion, and history: chronic quiescent process at right apex and a chronic active process with softening throughout upper lobe on the left side.

The cases which I have cited illustrate how the condition of the muscles in early, moderately advanced and far advanced cases of tuberculosis reflect the conditions within the lung and show how important it is for the examiner to acquaint himself with these changes

so that when he sees them in a patient his attention will be at once called to the probability of an intrathoracic inflammation, and be impressed with the necessity of a chest examination. The importance of this is emphasized by a study of Fig. 7.

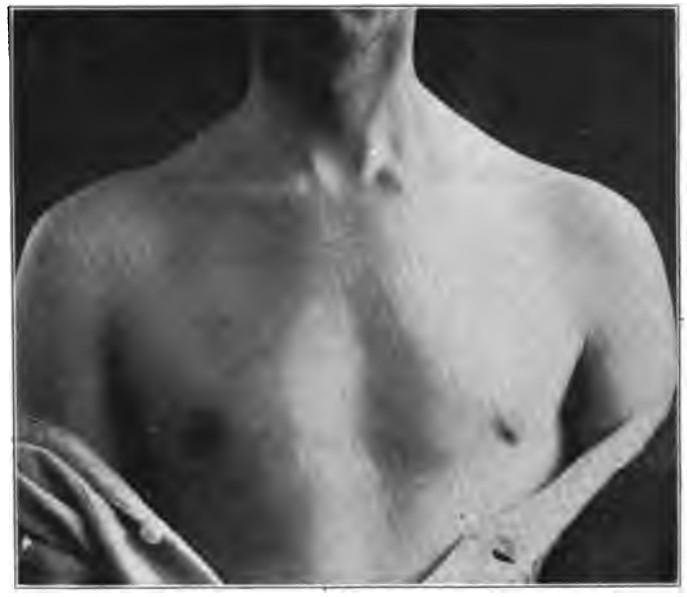

Fig. 7.—Illustrating both spasm and degeneration as observed clinically. This patient had suffered from a chronic tuberculous lesion occupying the upper part of his right lung, which is at the present time slightly active. The wasting of the subcutaneous tissue and muscles is shown by an increased flattening of the right chest, which amounts almost to a dishing out of the soft tissues, from immediately above the nipple to the clavicle, and a sinking of the supra and infra clavicular notches. It will also be noticed that the shoulder muscles on the right side are distinctly smaller than on the left. While the left is quite full the right appears much smaller, the substance having wasted. Owing to the difficulty of photographing, the right sternocleidomastoid fails to show the amount of spasm that is present. This muscle stands out slightly prominent on the patient. On the left side there has been a recent extension of the disease. This is shown very well by the condition of the neck muscles. Both the sternal and clavicular portions of the sternocleidomastoid stand out more prominently and are firmer than they should normally be, and the belly of the muscle is also somewhat enlarged. The shoulder muscles appear quite full.

On *palpation* the soft parts on the right side are thin and the muscles appear doughy to the touch. On the left side the muscles are firmer and offer a resistance much greater than normal.

In order to make my meaning clear to the reader I have slightly exaggerated the dishing on the right side in the picture, although unilateral wasting to the degree shown in this picture is occasionally found.

CHAPTER IV

Effects of Muscle Spasm and Degeneration

I shall now discuss the muscle change as it affects the patient on inspection, under which the posture of the head and the form of the chest will be especially considered, giving attention to lagging and the anomalies of the upper aperture of the thorax which are noted in tuberculosis.

The prominence of the sternocleidomastoideus and scaleni has long been recognized in advanced tuberculosis, but has been thought to be due to the general wasting of the disease. The posture of the head, when one studies it carefully, is not less striking than the prominence of these muscles, and goes to show that muscular contraction is an important factor in its causation. In order to understand the various postures and the muscles concerned in their causation, I will insert the following table giving the important neck and chest muscles, with their nerve supply and action.

IMPORTANT NECK AND CHEST MUSCLES, THEIR ACTION AND NERVE SUPPLY.

Sternocleidomastoideus.	When both act, depress head upon neck and neck upon chin; singly, flexes head; and, combined with splenius and obliquus inferior capitis of opposite side, draws head toward shoulder of same side; if head fixed, elevate thorax in forced inspiration.	Spinal accessory and deep branches of cervical plexus.
Scalenius anticus, medius and posticus.	Direct antagonists of muscles of back of neck; flex and rotate head; fixed below, turn spinal column to side; both sides together, bend spine forward; fixed above, elevate first and second rib.	Anterior branches of lower cervical nerves before forming the brachial plexus.
Rectus lateralis.	Acting on one side, bends head to side.	Suboccipital and deep internal branches of cervical plexus.
Trapezius.	Head fixed, elevates shoulder; shoulder fixed both acting draw head back; one acting draws head to same side.	Spinal accessory and branches from anterior division of 3rd and 4th cervical.
Levator anguli scapulæ.	Shoulder fixed, inclines head to same side and rotates backward.	Inferior division of 3rd and 4th cervical.
Rhomboidei major and minor.	Acting with trapezius, draws scapula to spine.	Branches from 5th cervical.

Latissimus dorsi.	Arm fixed, raises lower ribs.	Long subscapular (5th and 6th cervical).
Serratus posticus superior.	Superior elevates ribs.	Superior by external branches of posterior division cervical nerves; inferior by external branches of posterior division of lower dorsal.
Inferior.	Inferior draws lower ribs downward and backward.	
Pectoralis major.	When arm fixed, elevates ribs and expands chest.	Anterior thoracic (5th, 6th and 7th cervical).
Diaphragm.	Enlarges thoracic cavity from above downward antero-posteriorly and laterally.	Phrenic from 3rd and 4th or 4th and 5th cervical and branches of the intercostales from the VII to XII.

From the above table it is very plain, granting that my hypothesis that the segmental disturbance of the motor nerves follows the same or similar laws to those of the sensory, as worked out by Head, that an inflammation of the apex of a lung will show itself in a reflex spasm of the neck muscles, causing them to assume a contraction of a tonic character. Not only are the superficial muscles involved but the deep ones as well. If the irritation continues for a long time, the muscles degenerate and with the degeneration comes shortening. So, the posture of the head and the conditions about the neck differ according to whether the process in the lung is a recent or old one.

In unilateral acute primary inflammations of the lung, the posture of the head is unchanged from the normal, but the muscles usually stand out more prominently than their fellows. Through the constant muscle tension, however, the head, after a time, assumes a new posture as previously mentioned. Whether this occurs more as the result of the spasm or of the degeneration, I do not know; but I do not doubt that both are factors. The bending of the head to one side is a very common occurrence in chronic pulmonary inflammations. Another posture sometimes found is that resulting from contraction of one sternocleidomastoideus, the chin turning to the opposite side. Where both lungs are involved, both sternocleidomastoidei contract and, if the thorax be fixed, they at times draw the head and neck forward, giving a decided curve to the neck and shoulders, causing round shoulders as mentioned above.

It must be remembered in this connection that a bending of the head to one or the other side is not an uncommon condition in individuals who are well; and, such a position of the head must not necessarily be considered as a sign of pulmonary involvement. But if the

examiner will take the pains to have a number of patients suffering from advanced pulmonary tuberculosis stand or sit in a row, and then interest them so that their heads assume their natural positions, most of them will incline toward the side of the most severe and most extensive involvement. In determining the side to which the head bends, great caution must be used in order to avoid error; for people sitting down in ordinary positions, bend the head first to one side and then to the other. But, if the examiner will seat himself directly in

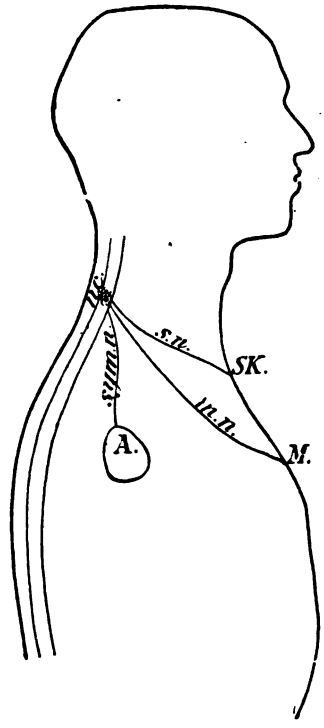

Fig. 8.—Showing the path of the reflex stimulation from the inflamed viscera *A.* through the Sympathetic *Sy.n.* to the cord where the irritation causes disturbance of the adjacent cells in the same segment of the cord *n.c.*, and reflects the stimulus through the motor roots *m.n.* of the anterior horn to the muscle *M.* causing contraction and later degeneration, and through the sensory roots *s.n.* of the posterior horn to the skin *SK.* causing disturbances in sensation.

front of the patient and engage him in earnest conversation for a short time, he will usually note that, if the patient has a lesion at one apex which has existed for a long time, long enough for degeneration of the muscles to ensue, the head will naturally be carried to that side. It is possible that occupational postures sometimes cause the same thing and possibly, at times, controvert it; but I have observed this sufficiently often, in cases where I could rule these factors out, to make it a sign well worth some attention in physical diagnosis. In advanced

cases where both lungs are involved it often furnishes a clue to the side which was first seriously involved.

The condition of the neck muscles in the presence of active disease in the apex is so very striking that it is difficult for me to see how we have failed to recognize it sooner. For those who have had little or no experience in the eliciting of these muscle changes I think it is easiest to detect them as they affect the sternocleidomastoidei and scaleni; but, when one has become thoroughly familiar with the changes, the trapezius, levator anguli scapulæ, rhomboidei, and pectorales also show it plainly. Fig. 8, p. 51, shows the path of the reflex.

That the muscle change is a factor in changing the type of respiration and in altering the form of the thorax there can be no doubt. This is much more evident if we accept the newer theory of respiration, as described by Keith (37), thus: "A consideration of the evidence now at one's disposal makes it difficult to believe that normal expiration is merely the result of an elastic recoil. The recoil is evidently under muscular control. The antagonist of the diaphragm is the musculature of the belly wall; it is difficult to believe that the replacement of the diaphragm and the abdominal viscera at the end of an inspiration is merely an elastic recoil of the abdominal musculature." Further attention to Keith's idea of respiration and further quotations from his writings will be given under the discussion of Freund's theory of ankylosis of the first rib.

With his conception of the respiratory acts we must look upon the respiratory motion of the thorax as emanating from two fixed bases, one the anterior neck muscles with their attachments to the cervical vertebræ and skull and the other the abdominal muscles with their attachments to the bony pelvis. Inspiration is a wave starting from above and traveling downward, while expiration is a wave beginning below and traveling upward. This explanation throws light on the functions of the intercostal muscles and shows that they are probably both inspiratory and expiratory in their action, depending upon whether they are pulling from above or below. Strengthening this view Keith quotes approvingly the observation of Sibson (38) that the tenth and eleventh intercostal spaces widen during inspiration and contract during expiration.

To comprehend fully the muscular acts of respiration we must consider both the action of the individual muscles and the muscles as a whole. If the head and neck are fixed, contraction of the sternocleido-

mastoidei elevates the thorax. These muscles are brought into play in forced inspiration. The scaleni contracting, with the neck fixed, elevate the first and second ribs, thus acting as inspiratory muscles. The intercostales acting with the first rib as a base, elevate the ribs which support their lower attachments and act as inspiratory muscles. The pectoral muscles, when the arms are fixed, help to elevate and expand the chest. The contraction of the diaphragm forces the abdominal viscera downward and forward, pushes the abdominal walls outward and at the same time enlarges the thorax from above downward. The contraction of the flat muscles of the abdomen, oblique transversalis, and rectus when the pelvis is fixed, depress the thorax, hence serve as muscles of expiration. This can be understood by a study of figures 9 and 12 (pp. 54 and 61). When the lower ribs are thus fixed by the abdominal muscles and made a basis of muscular action, contraction of the intercostales act as expiratory muscles, by depressing the ribs to which they are attached above.

The individual action of the principal muscles and groups of muscles which are brought into play in respiration has just been described. This, however, does not give us a comprehensive understanding of the respiratory act, for it is a composite action resulting from the combined contraction of many muscles. The inspiratory effort is spent in elevating the ribs, forcing the abdominal viscera down into the abdomen, thus decreasing the area in the abdomen and increasing the area of the chest from above downward. The expiratory effort, on the other hand, is spent in restoring the abdominal area and decreasing the diameter of the chest, and at the same time increasing the antero-posterior as well as the lateral diameters of the thorax. Taken as a whole, we must consider inspiration as a muscular contraction starting with the neck and first rib as a base and expiration as a muscular contraction starting with the pelvis and lower ribs as a fixed base. Of course it must not be forgotten that the diaphragm is the chief muscle of respiration and that the chest muscles are secondary.

With this conception of respiration and our recognition of the occurrence of spasm and degeneration of the chest muscles as a result of intrapulmonary inflammations, both of which have a tendency to shorten the muscles, we can explain more satisfactory than heretofore the symptoms of lagging and diminished motion, and the contractions of the chest wall which result from intrathoracic diseases.

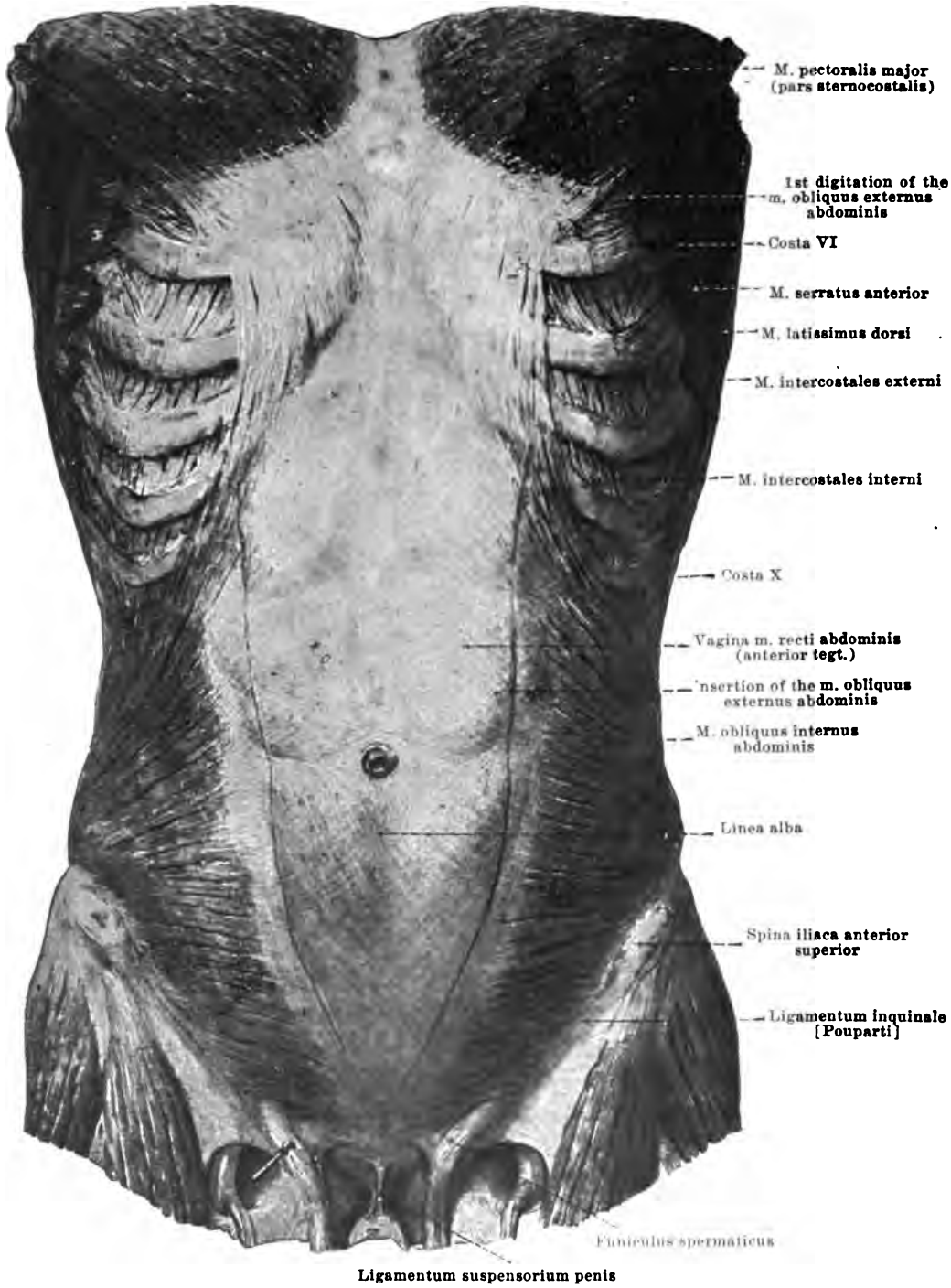

Fig. 9.—Muscles of the abdomen (second layer) viewed from in front. Contraction of the diaphragm in inspiration encroaches on the abdominal cavity from above, forces the abdominal viscera downward and forward, putting the abdominal muscles, recti, obliqui, and transversales on the stretch. It can be seen that their contraction must be an important factor in replacing the diaphragm, restoring the abdominal cavity and diminishing the thoracic cavity; in other words in expiration.

CHAPTER V

Cause of Lagging as a Sign of Apical Involvement

Lagging is a very important early sign in tuberculosis. It is recognized by clinicians as being very suggestive of apical disease. It is elicited by either inspection or palpation, the latter being easier and more reliable. If the examiner, standing behind the patient who is seated on a stool, will place his hands, with his thumbs in the supraspinous fossæ and his fingers extending down over the front of the chest, he will detect lagging of the apex, if it is present, by the respiratory excursion being either diminished or slower than normal in starting.

Lagging has been accounted for heretofore as being due to the fact that the infiltration in the lung interferes with normal pulmonary elasticity, thus preventing normal expansion. This, no doubt, is a factor to be considered; but this symptom is often present very early when it seems difficult to believe that there is enough infiltration to interfere with the elasticity of the lung sufficiently to explain it. When we consider, however, that there is a tonic contraction of the scaleni and sternocleidomastoideus muscles on the affected side as a very early sign of apical disease and that the result of a contraction of these muscles is to raise and fix the sternum and limit the motion of the first and second ribs on the affected side, it can be seen at once that this favors a disparity of the respiratory movement between the well and the affected side. The affected side appears to lag behind the well side in the point of time of beginning inspiration, and the total respiratory excursion on the affected side is lessened to a greater or lesser degree because the muscle spasm interferes with the free movement of the chest wall. When we add to the action of the scaleni and sternocleidomastoideus, the disturbance in the motion of the diaphragm which occurs in early tuberculous infiltrations, and recall the effect of such limited action upon the motion of the thoracic wall, we are led to believe that muscle change of reflex origin is the principal factor in the production of "lagging." I am further strengthened in this view and particularly in the part played by the diaphragm in its

causation, by recent careful observations of the relative excursion, not only of the apex but of the sides of the chest as a whole. To my surprise, I found that lagging in early tuberculosis is not confined to the apex, as usually described. Careful observation shows that the motion of the entire side is limited (39). This is what one should expect, if the diaphragm is an etiological factor, for the action of the diaphragm is exerted on the lung as a whole. As it descends, it increases the superoinferior diameter of the lung, also widens the lower thoracic aperture, thus enlarging the lung in all its axes. Therefore, the limited motion of the side of the chest, as a whole, is a sign of early pulmonary involvement as well as the long-described and well-recognized lagging of the apex; and I would suggest that if the examiner will but strip his patient to the waist and test the relative excursion of the two sides at the base of the lung, he will have valuable evidence bearing upon the intrathoracic condition. A lessened motion is very suggestive of inflammation within that side of the thorax. I have noticed that, where both lungs are the seat of disease (tuberculosis), an acute lesion, though relatively small, will produce greater lagging of the chest wall than a chronic inactive or less active lesion, though involving a much greater area. In this, the diaphragm seems to follow the same laws as the other muscles which take their nerve supply from the cervical portion of the cord, and I do not doubt that it undergoes the same spasm and degeneration; at least our clinical study of its action would indicate this. Krönig has partly appreciated this same fact. He has called attention to the fact that a lessened excursion of the lower edge of the lung may be detected in early tuberculosis.

CHAPTER VI

Cause of Flattening of the Chest Wall on the Affected Side

The cause of the flattening of the chest, which so often occurs in chronic pleurisy, empyema, and on the affected side in tuberculosis, and which often assumes a high degree when there is a marked destruction of lung tissue, is usually given as being due to the atmospheric pressure forcing the bony thorax to contract in order to occupy the space previously occupied by lung tissue. I formerly accepted this as the probable explanation (40).

That this is not a reasonable and sufficient explanation will readily appear when we consider the facts pertaining to these muscle changes and their influence upon the bony thorax. The thorax must be considered as one cavity. While it is divided into a right and left half by the mediastinum, yet this is a yielding partition and is quite readily movable. A loss of tissue on one side is readily compensated for by an increase in volume on the other side and a shifting on the part of the mediastinum. An increase of tissue on one side, or a pleural effusion or pneumothorax readily displaces the mediastinum toward the other side.

If compression of the ribs over the affected side were due solely to such a simple cause as atmospheric pressure, as is generally stated, we would expect the chest as a whole to be compressed instead of the part over the area which is the seat of loss of tissue; for the atmospheric pressure is equal on each and every portion of the chest wall. It would seem rational to expect the pliable organs of the thorax to readjust themselves rather than that the bony thorax in a certain part should be compressed to compensate for the loss of tissue.

We must look elsewhere, then, for a satisfactory explanation of chest contractions. There must be some strong local compressing force which accounts for the contraction being limited to the side or area affected. This force, we have in the pressure exerted by the muscles which are shortened, both when in spasm and when degenerated. This influence is the greater the more direct the pull, consequently we should expect to find it greater at the apex in early childhood when

the anterior portion of the first rib is on a higher plane than it is later in life. See Fig. 1 (p. 17). Pathological flattening of the chest being nearly always due to inflammatory diseases within the thoracic cavity, reflex interference with the normal motion of the diaphragm must be considered as a part of such flattenings.

To appreciate the part played by the chest muscles fully it is necessary to understand the position and direction of the force exerted by shortening of those muscles which, by their attachments to both the chest wall anteriorly and the spinal column and skull posteriorly, bridge over the upper aperture of the thorax. The most important of these muscles are the sternocleidomastoidei and the scaleni. See Figs. 2 and 10 (pp. 19 and 59). The former arises from two heads, one of which is attached to the sternum, and the other to the inner third of the clavicle. These two join and form a large muscle mass, which passes upward and backward to be inserted into the mastoid process. The pull, when this muscle is contracted, if the head is fixed, is upward and backward. If not only the head be fixed, but also the lower part of the thorax, then the backward pull is exaggerated.

The scaleni are shown in Fig. 10 (p. 59) and are three in number. The scalenus anticus arises from the upper surface of the first rib and is inserted into the anterior tubercles of the transverse processes of the 3rd, 4th, 5th, and 6th cervical vertebræ. The scalenus medius arises from the upper surface of the first rib and is inserted into the posterior tubercles of the transverse processes of the lower six cervical vertebræ. The scalenus posticus arises from the outer surface of the second rib and is inserted into the posterior tubercles of the transverse processes of the lower two or three cervical vertebræ. The pull of these muscles when the neck is fixed is therefore upward and backward, and when both the neck and base of the thorax are fixed the backward pull is exaggerated the same as with the sternocleidomastoidei.

Thus we can see that the tonic contraction and degeneration, which results in a permanent shortening of these muscles and which is produced reflexly from pulmonary inflammations, must have a tendency to shorten the antero-posterior diameter of the upper thoracic aperture.

The part played by the shortening of the muscles can readily be understood by referring to the action of the muscles of respiration on the living subject. The action of the scaleni may be understood by

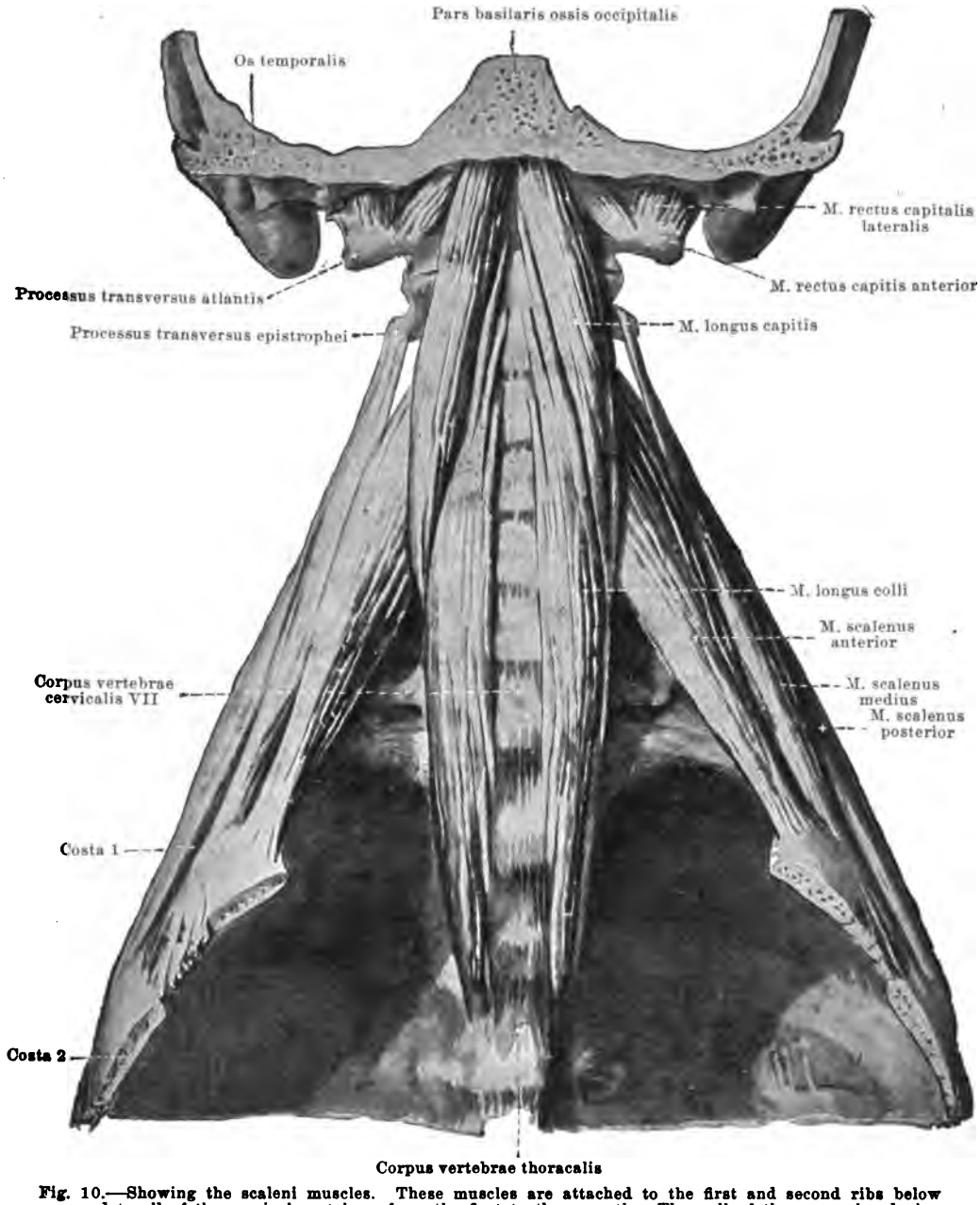

Fig. 10.—Showing the scaleni muscles. These muscles are attached to the first and second ribs below and to all of the cervical vertebrae from the first to the seventh. The pull of these muscles during contraction is upward and backward. In childhood (see Fig. 1), and in case the free action of the ribs is interfered with, the backward pull of the scaleni with their tendency to flatten the upper aperture of the chest is emphasized.

studying Fig. 10 and the action of the muscles of respiration as a whole are schematically shown in Fig. 11.

The inspiratory muscles as a whole work as a lever: the fulcrum being the lower ribs fixed by the abdominal muscles, the power is exerted from the cervical vertebræ and skull as a base through the scaleni and sternocleidomastoidei and the successive intercostal muscles; the

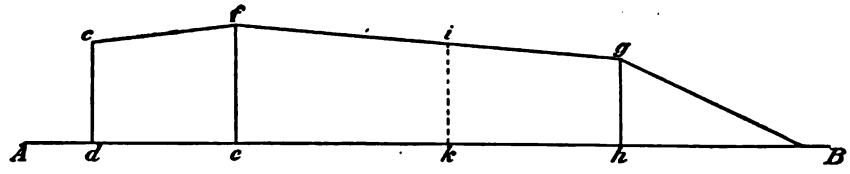

Fig. 11.—Represents the action of the muscles of respiration as a whole. *A.B.* the base line is the spinal column; *c.d.* represents the bony pelvis which furnishes a firm attachment for the muscles below; *f.e.* the lower aperture of the thorax; *c.f.g.B.* represents the muscles of respiration. The respiratory movements of the chest wall start from the upper aperture of the thorax *g.h.* with its attachments to the cervical spine *g.B.* as a base; or from the lower aperture of the bony thorax *f.e.* with its attachment to the pelvis *c.f.* as a base. Any shortening of the muscles between *f.* and *g.* or *g.* and *B.* shortens the distance from *f.* to *B.* and as a result shortens the distance *i.k.* which represents the anterior posterior diameter of the thorax. Any shortening of the muscles between *c.* and *f.* or *f.* and *g.* also shortens the distance *i.k.* Spasm of the neck muscles and upper intercostales especially shorten the line *f.g.B.* in its upper portion, consequently it causes the greatest shortening of *i.k.* in the upper part of the bony thorax, causing flattening of the upper thorax, while spasm of the lower intercostales, diaphragm and abdominal muscles such as occurs in pleurisy at the base or in other lesions of the base of the lungs, causes the greatest shortening between *c.* and *g.*, producing shortening of *i.k.* at the base and flattening of the base of the thorax.

weight is the chest wall which is compressed by the pull which shortens the line between the lower ribs and the attachment of the neck muscles to the spine and skull.

This offers a rational explanation of the flattening of the upper aperture of the chest when the apex has been the seat of an old lesion. We often see this clinically in healed tuberculosis of the apex. The upper ribs having yielded to the constant muscular pull are depressed; the clavicle stands out unduly prominent, the supraclavicular and infraclavicular notches are exaggerated. This process may begin at the upper aperture of the thorax; but if one set of intercostales after another are reflexly thrown into spasm and later undergo degeneration, and if at the same time, there be a diminution of the elasticity of the underlying lung tissue, or, if its tissue gradually becomes destroyed, as we often see it in the upper lobes of the lungs in advanced tuberculosis, then a greater or lesser portion of the entire side of the thorax may flatten. This flattening may effect both sides of the chest as well as one, but, as a rule, it is found to the greatest degree on one side.

The localized contractions which are commonly found over the lower ribs in patients who have suffered from pleurisy or empyema are probably to be explained at least in part in a different manner. In such cases the diaphragm, lung, and costal pleura may adhere together or

the diaphragmatic and costal pleura may become adherent, obliterating the complemental space. Under such conditions, when the diaphragm contracts, it cannot strip itself from the costal pleura, consequently it exerts a pull upon the ribs. This pull, being more or less constant, causes the ribs to yield, and after a time draws them in, producing the localized flattening. This is illustrated by Fig. 12 (a) and (b), (a) showing the physiological action of the diaphragm and chest wall and

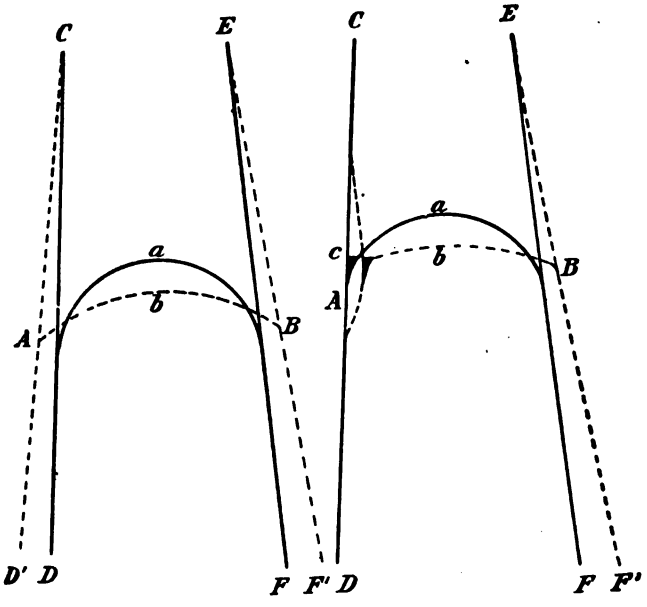

Fig. 12.—(a) Schematic representation of the contraction of the diaphragm showing the enlargement of the thoracic cavity from above downward, the diminishing of abdominal cavity from above downward and widening of the lower thoracic aperture. *A.B.* is the diaphragm and *C.D.* and *E.F.* are the thoracic walls on expiration. When *A.B.* contracts on inspiration it shortens and changes position from *A.a.B.* to *A.b.B.* This same action forces the abdominal viscera downward and forward and this in turn increases the antero-posterior and lateral diameters of the thorax. *C.D.* takes the position *C.D'.* and *E.F.* takes the position *E.F'.*

Fig. 12.—(b) Represents schematically the action of the diaphragm in the production of localized flattening when the complemental space has been obliterated by inflammatory adhesions. *A.B.* is the diaphragm and *C.D.* and *E.F.* are the thoracic walls on expiration. The complemental space *A.c.* is obliterated by adhesions. When the diaphragm contracts it is no longer able to strip itself from the ribs owing to the adhesions from *A.* to *c.* As a result of this the free movement of the diaphragm represented by the normal distance *A.a.B.* is shortened and represented by the distance *c.b.B.* Contraction of the diaphragm then, instead of stripping itself from the costal surface draws on the ribs and pulls them in the direction of *c.b.B.* thus favoring local flattening.

(b) showing the pathological action when adhesions of the pleura at the base have occurred.

The flattening in empyema and chronic pleurisy has been suggested to be due to the fact that the visceral and parietal pleural surfaces are adherent and that fibrous bands connect the surface of the lung with the root of the lung and exert a pull at each inspiratory act. It seems to me, however, that this explanation is not sufficient.

When we have a unilateral flattening affecting the entire lower por-

tion of the thorax, as occurs in old pleurisy, empyema, and tuberculosis, besides the local adhesions of the diaphragmatic, costal, and pulmonary pleura mentioned above, we have the same principles that operate in flattening the upper portion of the chest, as seen in Fig. 11. The motion of the diaphragm is limited. The lower intercostales are in contraction. The length of the line from the pelvis to the first rib is shortened. The pressure, however, instead of being exerted to depress the upper portion of the chest, is exerted over the lower ribs and depresses them, causing a flattening of the side. This flattening is especially favored by the restricted action of the diaphragm, because the diaphragm has a piston-like action and when it contracts normally it forces the abdominal viscera downward and forward, lessening the size of the abdominal cavity in the vertical direction, as seen in Fig. 13, explaining the contraction of the diaphragm. To compensate for this, the muscular portions of the abdominal wall yield, and in this manner, the antero-posterior and the transverse diameters are increased. The same action forces the lower ribs outward, increasing the size of the lower aperture of the thorax. It can be seen, then, that when the motion of the diaphragm is interfered with, as it is in empyema, pleurisy, and pulmonary diseases, especially those involving the bases of the lungs, the force which causes the widening of the lower costal arch is lessened and if this interference continues long, as it does when these diseases become chronic, there is a prolonged absence of the force which widens the lower costal arch. This lessened action of the diaphragm is for the most part confined to the side involved. When we add to this the fact that the costal and diaphragmatic pleural surfaces are sometimes adherent, as mentioned above, and that the intercostales, and at times the abdominal muscles, are shortened by spasm on the side of the involvement, especially in the presence of pleurisy involving the lower costal and diaphragmatic portions of the pleura, we can understand that three important conditions are present which favor flattening of the lower portion of the chest wall: a lessening of the force which expands it, a retraction of the wall by the diaphragmatic adhesions, and a shortening of the muscles between the pelvis and the first rib. In advanced tuberculosis this is often counteracted by an emphysematous condition; the respiratory area of the upper portion of the lungs being diminished, the lower lobes take upon themselves a state of compensatory emphy-

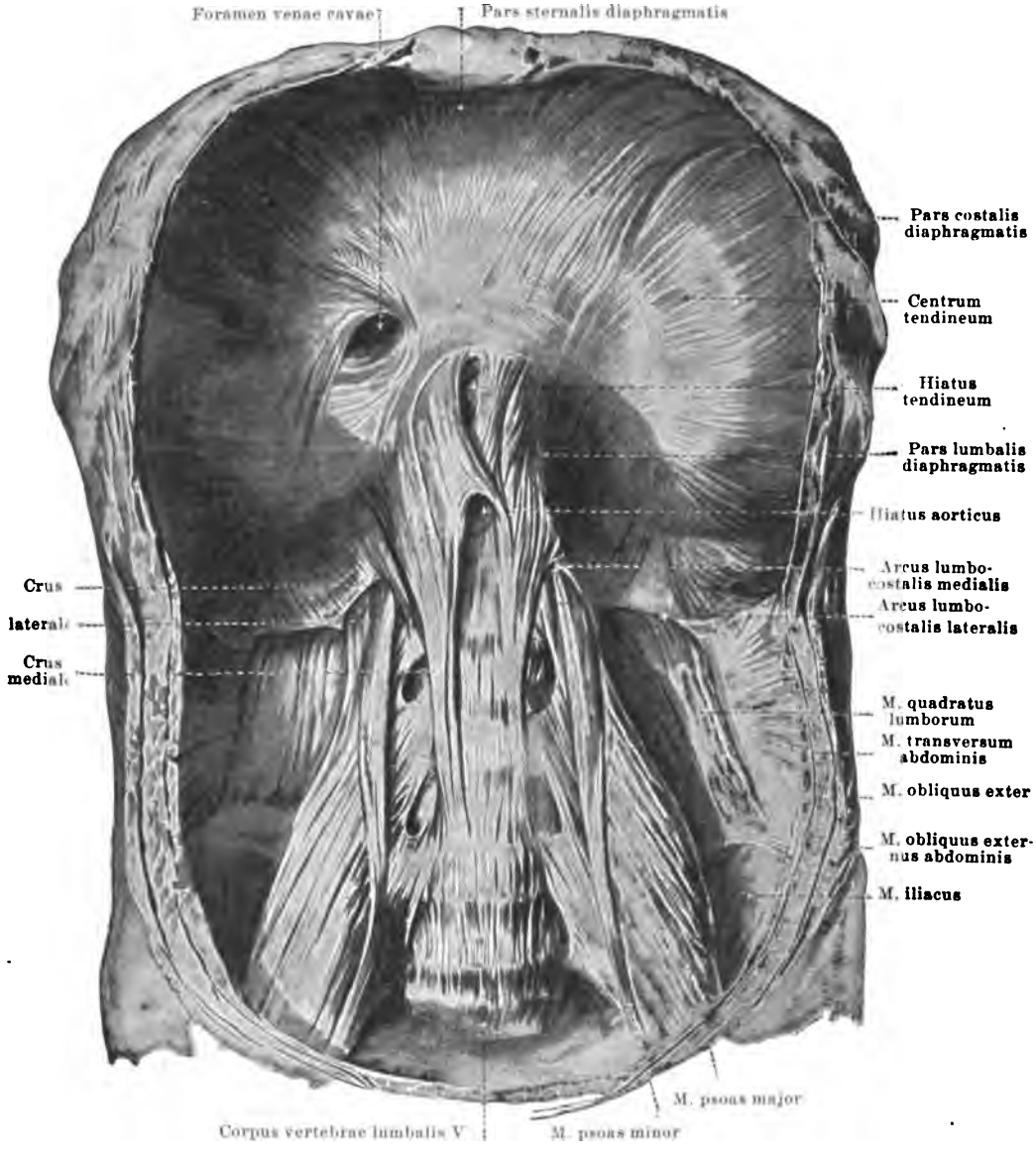

Fig. 13.—Diaphragm viewed from below and in front. The diaphragm is made up of central fleshy tendons (pars lumbalis) and thinner fibers running to the ribs (pars costalis) and sternum (pars sternalis). The contraction of the diaphragm consists in shortening of both the crura (pars lumbalis) and the pars costalis and pars sternalis. With the abdominal viscera as a fulcrum, the contraction of the diaphragm widens the lower portion of the thorax as shown in Fig. 12 (a). In inflammations of the lungs its motion is restricted. It is probably in tonic contraction for the same reason that the neck and chest muscles are in contraction, having its nerve supply in part from the cervical portion of the cord. This restricted action seems to be confined to the side involved. In some cases the limited motion does not seem to be present, but where it is present there is probably an actual shortening of the fibers, the same as in the surface muscles when they are contracted.

sema, which widens the lower ribs instead of allowing them to yield to the compressing force.

One may study the lessened action of the diaphragm and the compressing force of the abdominal and lower intercostal muscles in the production of flattening by observing the lessened bulging of the abdominal wall, the lessened descent of the abdominal viscera, and the lessened motion of the lower ribs in unilateral inflammations of the lungs, particularly of the bases and the pleura covering them.

Hilton, in his classical lectures on rest and pain, calls attention to the influence of pleurisy in causing contraction of the abdominal muscles as well as in the production of pain, in the following words: "In cases of costal pleurisy of the lower half of the chest, it is a common occurrence that the upper half of the abdominal parietes is tightly drawn backward and the skin over that part very tender to the touch; and that pain, depending upon pleurisy of the lower half of the chest, is referred to this spot."

The lessened motion of the diaphragm in tuberculosis is the cause of a very important pathological condition, to which I would call attention briefly, even though not strictly belonging to my subject. The relationship between the action of the diaphragm and the circulation is not fully appreciated. Not only normal respiration but normal circulation depends upon the free action of the diaphragm. By the normal descent of the diaphragm the size of the abdominal cavity is decreased, and the tension within it increased, while the intrathoracic space is increased and its tension decreased. There should be a natural flow of blood from the abdomen into the thorax at each inspiration. Any factor, then, such as inflammations within the thorax, which limits the motion of the diaphragm, interferes with this normal respiratory movement and hinders the flow of blood toward the right heart and favors the congestion of blood in the veins. This is especially noticeable in the abdominal veins and accounts in part for the passive congestion of the abdominal viscera in tuberculosis. As mentioned in another paper (41), while there is a venous congestion of the abdominal viscera in all cases where the action of the diaphragm is interfered with, there is also a relative arterial anemia throughout the body. This furnishes an explanation of the clinical observation that patients suffering from tuberculosis look pale, while blood tests show a high percentage of hemoglobin and red corpuscles. Their paleness is due to the relative arterial anemia. The relative small amount of

blood in the arteries also affords another explanation of hypotension in tuberculosis. Again, it furnishes the conditions which make digestive disturbances common and difficult to handle, namely, a constant venous congestion of the abdominal viscera. Indigestion, enteritis, colitis, constipation, and flatulence are found in some degree in practically every case of tuberculosis that has extended beyond the early stage. The chronic congestion present not only favors such conditions but makes them difficult to treat successfully.

While tuberculous patients do not all suffer from enteroptosis, yet they suffer from the same trend of symptoms. Wenckebach (42) has described this condition well and I quote his words: "Studies in the anatomy and physiology of the diaphragm have shown that the normal action of the diaphragm and abdominal wall is a very important factor in favoring the flow of the venous blood from the abdomen to the heart. Enteroptosis, on the other hand, is a pathological condition affecting the function of the diaphragm and abdominal wall, which, almost invariably, produces disturbances in the venous return flow from the abdominal cavity to the heart. When one begins to observe he will find many cases of disturbances of this kind even without careful analysis of the activity of the heart and circulatory system. The most prominent symptoms are those of arterial anemia. The patient becomes tired quickly, has a feeling of dizziness which amounts at times to a true lipothymia. The skin is relaxed, the eye is hollow, the face pale, but not without a trace of cachexia. The pulse is small and frequent, but not always soft, and the extremities are cold. Oftentimes it is evident that the heart is supplying too little blood to the arterial system, nevertheless the heart tones are normal and the blood itself is of good quality. If there is neither disease of the heart nor true anemia present, where must we find the blood when the arteries are not well supplied with it? At times, as in some of the cases cited, one sees a general overfilling of the veins, at times even a slight edema and cyanosis. Sometimes, however, neither of these are present. The symptoms are such, however, that one is forced to believe that there is an increase in the amount of blood stored up in the abdominal vessels." Eppinger (43) has recently given us a very valuable monograph dealing with the diaphragm and its action, in which this same subject is well discussed.

CHAPTER VII

The Relation of Muscle Change to the Anomalies of the Upper Aperture of the Thorax

Our knowledge of the anomalies of the upper aperture of the thorax is largely due to the work of Freund, Hart and Rothschild (1, 2, 3, 4, 5, and 6). After an exhaustive study, these observers have arrived at the conclusion that an ossification and shortening of the first costal cartilage and a diminution in the movement of the manubriosternal articulation interfere with the free expansion of the apex, and are predisposing factors in the causation of tuberculosis.

Freund (1) observed the same contraction of the muscles of the upper aperture of the thorax that I have described, but accounted for the condition in a different manner. He looked upon it as a hypertrophy from overwork, caused by the muscles pulling against the ankylosed rib. He described it as follows: "In many patients who are suffering from beginning chronic tuberculosis of the apex of the lungs, one finds pathological changes, especially in the first costal cartilage, this change being an ossification. The bony change does not proceed from an inflammation of the pleura, for it usually begins first on the edges, then on the outer surface of the rib, and at last on the inner surface, and all takes place without the pleura being changed. This also occurs in beginning tuberculosis where the pleura is not yet involved. *Under these conditions one always sees strong antagonistic development of the scaleni whose point of insertion on the rib often shows an exceptional size.*"

Hart in his discussion of my theory, maintained with Freund that the contraction of the muscles observed by me was unquestionably due to overdevelopment of the muscle from pulling against the ankylosed rib. If this theory were correct, the overdevelopment would necessarily be confined to the scaleni, but this is not true. We find the same in the sternocleidomastoidei, pectorales, trapezei, levator anguli scapulæ, rhomboidei, intercostales, in fact in all muscles of the neck and chest; so it would seem that this of itself is proof sufficient to show that the cause must be other than that suggested by Freund and Hart.

My theory of one common cause for the spasm of all these various muscles, and the later degeneration of the same, seems more rational, especially since it is borne out by anatomical facts.

When Freund noticed the common coincidence of bony ankylosis of the first costosternal joint and apical tuberculosis, it was natural to inquire into the relationship of the two. As a result of his study he came to the conclusion that the bony ankylosis was a predisposing cause of tuberculosis. It must be remembered that the nature of tuberculosis was poorly understood at that time. The specific tubercle bacillus was not discovered until twenty years afterward. The clinical diagnosis of the disease had made but little progress and its pathology had not been worked out. Under these circumstances, it is but natural that Freund should have suggested the change in the thorax as being the cause rather than the result of tuberculosis. Without understanding that it was a specific disease, it was natural to explain it on mechanical grounds, and even without knowledge of its etiology, the mechanical conditions offer an attractive reason why the apices seem to be predisposed to infection. With our knowledge of the compensatory changes which occur in the intrathoracic organs, however, and with our knowledge of the changes that occur in the bony thorax as a result of tuberculosis, we are forced to examine carefully before committing ourselves to Freund's theory; although we must accept it as a splendid work for the time when it was produced. After being unnoticed for half a century, Freund reiterated his theory in 1901 (3).

Rothschild (6) has suggested that a limitation or absence of the sternomanubrial movement is a predisposing cause of tuberculosis. This theory is closely bound to the one suggested by Freund and according to our present knowledge must also be looked upon as a result rather than a cause. I am pleased to be able to support this view by so careful and able an observer as Keith (37), and will quote from his writings at length:

"The first rib has always been treated as merely one of the costal series. Its articulation to the spine, its ligaments, its muscles, its shape, its costal cartilage, its intimate union with the manubrium sterni, differ so markedly from the corresponding features of other ribs, that were only the anatomical evidence available, one would conclude that it differed from all the others in its respiratory function. An examination of its movements and the part it plays in expanding the lungs shows

this is so. The first pair of ribs and the manubrium sterni are bound intimately together by the broad and first pair of costal cartilages, and form, with the manubrium, a united structure which may be described as the lid or operculum of the thorax. See Fig. 14. Behind, this lid is articulated to the spinal column by a joint which is set more transversely and is wider in the extent of its attachments than any other of the costal arcs; in front, the lid is articulated with the body of the sternum at the manubriosternal joint. The manubriosternal joint must be counted amongst the important respiratory joints. Ankylosis of this joint is rarely seen before the fiftieth year, and it is uncom-

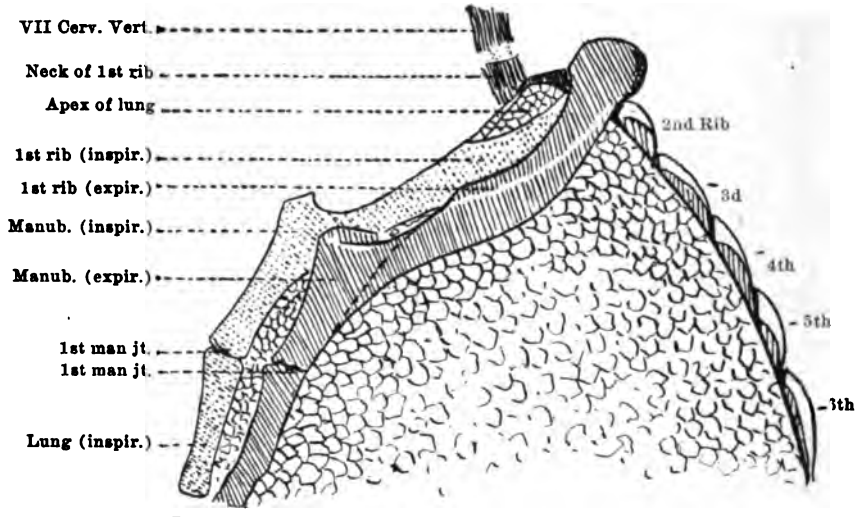

Fig. 14.—Showing the action of the first pair of ribs and manubrium.

mon before the sixtieth. The respiratory movement which occurs at it varies with the individual, with the type of respiration—being greater in those with the thoracic type—and with the extent of the respiratory movement. Braune estimated its movement at 5° to 13°; Rothschild, who regards limitation in the movement of this joint as a cause of consumption, estimated the average movement (in full inspiration) to be 15.85° in the male and 12.85° in the female; while the writer, who was unaware of Rothschild's observations, found it varied from 1° to 16°. The degree of movement depends chiefly on the inspiratory behavior of the body of the sternum, which is extremely variable. In some individuals the lower end of the sternum during the elevation of the thorax during inspiration may be drawn toward the spine or move forward to a less degree than the upper end of the body of the sternum; in such the sternomanubrial movement is free. If, on the other

hand, the lower end of the sternum moves more freely away from the spine than the upper end, the movement at the joint is less extensive. At the sternomanubrial joint, the operculum or lid of the thorax articulates with the anterior thoracic wall. The prominence of this joint on the surface of the thorax is extremely variable—so many conditions may render it unduly prominent. Ludwig, the Parisian physician, is said to have regarded an undue prominence of the articulation (Angulus Ludovici) as an indication of phthisis; but researches made by recent German writers have failed to trace such a statement in Ludwig's writings. Rothschild found that the sternomanubrial movements were limited or absent in phthisical subjects, and ascribed the susceptibility of the apex to phthisis as due to an ankylosis or limitation of movement at this joint. In his opinion a free manubrial sternal movement is necessary if the apex of the lung is to be properly expanded. Freund attributes the incomplete expansion of the apex in the phthisical action to a congenital shortening and ossification of the cartilage of the first rib. It is true that the necks of the first pair of ribs are so articulated to the spine that with the elevation of the manubrium sterni there is some degree of torsion in their cartilages, but the amount of the torsion is slight in extent because of the particularly loose manner in which the heads of those ribs are bound to the first dorsal vertebra. The writer, unaware of the observations and theories of Freund, and Rothschild, had concluded that the ossification of the first costal cartilage and limitation of the sternomanubrial movements were consequences rather than causes of a limited expansion of the apices of the lungs. In dealing with the significance of the division of the lungs into lobes, the direct influence of the diaphragm on the apices of the lungs has been already pointed out. The upward movement of the first pair of ribs and manubrium expand chiefly the anterior or ventrolateral part of the apex of the lung; the movement has but an indirect influence on the dorsal part of the apex, especially that part lying in front of the necks of the first and second pair of ribs. It is the dorsal part of the apex of the lung that is the common initial site of pulmonary tuberculosis. To secure a free expansion of that area of the lung a full diaphragmatic contraction is much more effective than any movement of the upper thorax."

From my discussion it can readily be seen that the muscle change described by Freund in 1859 as an hypertrophy due to overwork, being caused by the muscle pulling against an ankylosed rib, is more likely

a contraction of the muscle caused by the inflammation within the lung acting reflexly through the cord. It is also probable that this contraction of the muscles covering the apex, together with the limited motion on the part of the diaphragm which is present in even small pulmonary lesions, together with the decreased expansibility and lessened elasticity of the parenchyma of the underlying lung, caused by the inflammatory process within, are causes of lessened motion at the apex; and that these conditions, together with the trophic changes which occur in the bone and cartilage as a result of the reflex stimulation of the nerves which supply these structures, favor ankylosis and ossification; for it is extremely probable that the nerves supplying the costal cartilage and the costosternal and manubriosternal joints are also irritated reflexly from the intrapulmonary inflammation, the same as those supplying the skin and muscles, for, as Piersol says, "a similar arrangement of the nerves is seen in the joints, where the same nerves supply the skin covering the joint, the muscles which move it, and the joint structures." From this it seems probable that the cause of the ossification of the cartilage and the ankylosis of the costosternal and sternomanubrial articulations is also a reflex.

As another illustration of this same action upon the joints, I would cite dry arthritis, which is found so commonly affecting the shoulder joint when the lung is the seat of advanced tuberculosis. This condition is so common that it can be found in a great majority of cases. I have seen it in some instances so severe that a partial ankylosis was produced.

This has been considered to be of toxic origin; but, I believe this opinion is erroneous and would suggest that it is more probably due to changes in the nerve fibers supplying the joint causing trophic changes and producing a condition analogous to the atrophy found in the muscles.

Another phase of the theory supported by Freund is the shortening of the first rib. The action of both of these phenomena, described by Freund and Rothschild, is supposed to be that of interfering with the proper expansion and aeration of the lungs and indirectly retarding the blood and lymph flow to them, causing the apices to be points of lessened resistance. According to Keith's study of the mechanism of respiration, the lessening of the motion of the upper ribs and manubrium would not have as much influence in checking the free respiratory movements of the apex in a healthy lung as has been thought, and con-

sequently would not be a large contributing factor in the retardation of the blood and lymph flow and the causation of tuberculosis. When the disease is already present, however, the neck muscles are thrown into a state of contraction, and the movements of the diaphragm are lessened; the accordion action of the lungs is interfered with and the respiratory movements of the apex are diminished. A point against Freund's theory is the fact that the parts of the apex especially influenced by this compression are not the usual seats of the primary tuberculous infection. If Freund's theory were correct we would expect tuberculosis to begin on the anterior or inner surface of the apex, while in truth it usually begins, as pointed out by Birch Hirschfield (44), on the posterior aspect of the apex.

When one comes to study the relationship between the tuberculous foci found in the apices of the lungs and the anomalies found in the upper aperture of the thorax, he is confronted with a mass of evidence which points to the tuberculous infection being the cause rather than the effect of the bony and cartilaginous changes.

Since studying this question, I have had the opportunity of examining many young children whose parents were afflicted with tuberculosis. When the lung had already become infected and changes on physical examination were present, I could detect, almost without exception, not only a lagging of the apex which showed the involvement, but also a slight flattening of that portion of the chest which is on a level with the first rib, as compared with the same area on the healthy side. The muscles covering the apex were either in spasm or showed degeneration; and, as a rule, the side showed some degree of lagging indicating that the diaphragm was also involved. I have not found this same condition present in those children where I could rule out pulmonary infection. It seems to me, then, that the weight of argument is in favor of the pathological condition within the lung being the cause of the contraction of the neck muscles, thus interfering with the action of the diaphragm and the production of the limited motion of the side affected and finally the shortening of the antero-posterior diameter of the upper aperture of the thorax on the affected side. As the child grows and the chest increases in size, it can be readily understood that this shortening of the antero-posterior diameter will be emphasized, for the part affected will not enlarge as rapidly as the side unaffected; and, where it is further understood that the nerves supplying the rib, cartilage, and articulation are likewise in-

volved in the reflex mechanism, it can be seen that the nutrition of these parts must suffer.

The first rib is probably congenitally shortened in some instances. Rachitis must be looked upon as a cause in a certain proportion of cases. When tuberculosis develops in apices where the ribs were previously shortened from the foregoing causes, the conclusion might be erroneously drawn that the shortened rib stood in a causative relation. When we consider, however, that tuberculosis develops in chests which were previously well developed, and is able to produce marked changes in the bony contour of such chests; and, then, when we note the lessened motion and rigidity of the thorax often caused by this disease, we see that there is a possibility that the effect may have been taken for the cause in explaining the phenomena described by Rothschild and Freund. When we add to this the frequency with which tuberculosis of the apex is found in people who are not clinically tuberculous, we have further evidence to make us hesitate. Hart found apical tuberculosis in 63.4 per cent. of 400 post mortems. Babes found 74 of 100 children who died of diseases other than tuberculosis, to be infected with the disease. These statistics are in harmony with those of Nägeli (45) who found infections in 97 per cent. of bodies post mortem and with the clinical experiences of Hamburg (46) who found 94 per cent. of children in the St. Anne Children's Hospital in Vienna to be tuberculous, as determined by the tuberculin test.

There is a time when these infections, which in early years are confined for the most part to the bronchial and mediastinal glands, spread to other parts of the body, usually the apices of the lungs. Just when this occurs we do not know, but it usually occurs without the production of signs or symptoms which we have been able to recognize.

While we have not as yet been able to recognize clinically the changes that occur when the transfer of infection takes place, yet it is probably attended by important phenomena. I have shown how clinical tuberculosis is reflected upon the neck and surface of the thorax by spasm of the muscles. We find this muscle contraction present in our earliest cases of clinical tuberculosis and recognize it to be due to a stimulation caused by the inflammation in the lung which passes to the cord, causing an irritable condition of the adjacent cells in the same segment, from which the motor impulse arises, which expresses itself in a contraction of the surface muscle. Therefore we are forced to conclude that these muscle contractions occur as soon as

inflammation is present in the lung or as soon as it is able to cause a condition of the segment of the cord sufficiently irritable to produce reflex phenomena. We are also justified in assuming that the nerve supply of the first ribs and first costal cartilage is also early affected reflexly, thus favoring degenerative changes.

As previously mentioned, the neck muscles show the contraction first. The motion of the diaphragm is also interfered with early. We cannot fail to understand, then, the effect of a tonic contraction of such muscle as the scaleni, though it be only of slight degree, in altering the normal conditions surrounding the upper aperture of the thorax, especially when conditions are present which lessen the motion of the chest wall and lung within. The effect of such a muscle pull would be greater on children than on adults, and I can see how it would interfere with the proper motion of the upper portion of the chest, cause a shortening of the ribs, favor ankylosis of the costosternal articulation, and lessen the motion at or cause ankylosis of the manubriosternal joint. It helps us to understand the production of ossification and ankylosis when we remember that the trophic nerves supplying these cartilages and joints are subject to the same constant irritation as the muscles covering them. It is probable that the reflex stimulation of the nerve supplying the first rib and first costal cartilage by the inflammation in the lung in conjunction with the muscle changes which I have mentioned is a very important factor, especially in infections which occur in early life, in causing ossification of the first costal cartilage, ankylosis of the first costosternal articulation, and shortening of the first rib.

We can also see how such a shortening of the neck muscles might cause a twisting and bending of the cervical spine, this being a causative factor in the production of scoliosis.

That the tuberculous infection is a factor in the production of the shortening of the first rib can be supported by the statistics of Hart (4). While this anomaly is present in a large percentage of all cases of apical tuberculosis, yet, according to Hart's observations, it is far more common in those with a hereditary history. The probable reason for this is clear. Children who are born in tuberculous families come in contact with bacilli in greater numbers and more frequently than those who are not so directly exposed to infection; consequently they are more likely to be infected and also more liable to have the disease spread. The result is that the apex of the lung

probably becomes infected earlier in children of tuberculous than in those of nontuberculous families, and consequently causes the reflex stimulation of nerves supplying the muscles of the neck and of nerves supplying the first cartilage and first costosternal joint earlier, thus causing the changes in the first rib and upper aperture of the thorax to be more common and more pronounced than where the lung is infected later in life.

It would seem to me that it is possible to obtain very reliable information relative to the time when the infection passes from the glands to the lung by studying the upper aperture of the bony thorax. If the infection occurs in childhood while the chest is small and the bones are soft, an asymmetry is very prone to occur; while, on the other hand, if it occurs later in life, the ribs have already attained their full growth and a shortening of the rib or a shortening of the antero-posterior diameter of the upper aperture is not likely to occur. This is in harmony with the observation that flat-chested children are often found in tuberculous families, for here, as has been shown by Hart, the lungs are most apt to be affected during early life and when, according to the same observer, the anomalies of the upper aperture are most common. The pathological facts established by Hart to the effect that a large proportion of all tuberculous subjects show these anomalies of the upper aperture of the thorax is also in direct harmony with the recent teachings regarding tuberculous infections: viz, that tuberculosis is primarily a lymphatic disease, but that it spreads to the lungs from the glands, the transmission usually occurring in childhood.

It seems perfectly clear after carefully considering all of the phenomena attendant upon the changes in the upper aperture of the thorax that the ultimate relationship between them and tuberculous infections of the apex of the lung as suggested by Freund and Rothschild, must be reversed and that these anomalies, in so far as they are related to tuberculosis, are a result and not a cause.

CHAPTER VIII

Neck, Chest, and Shoulder Pains of Reflex Origin

While I do not wish to enter into a full discussion of the many pains that accompany intrathoracic inflammations (especially when found in tuberculosis), yet I cannot refrain from suggesting that their character indicates that some of them are phenomena of reflex origin (47). I am well aware that the theory of a reflex will not explain all, for we must recognize pleurisy, pressure pains, and general pains which are found in all parts of the body which may be of toxic origin. But, aside from these, we have regional or localized pains, the same as we have localized muscular contractions and atrophies, which must be explained by some locally acting cause. As a hint to explanation we find these localized pains for the most part, as far as I have been able to determine, on the same side on which the involvement exists. Thus it seems natural to suggest for them a common cause with other reflex phenomena.

Of such regional pains the vague sensations which are found in the shoulder or shoulders in case of infection of both lungs must be mentioned. These often begin as early as clinical symptoms occur. They vary in intensity from a mere "feeling of being tired" to an aching more or less severe. Aside from these vague feelings of discomfort we find pains of a very severe character which leave no doubt as to their nature. They are a true neuritis. These we find especially about the neck, shoulder (occasionally running out into the arm), and upper portion of the chest both anteriorly and posteriorly. I have seen some instances in which the pain in these cases was so severe that it was necessary to resort to morphine injections. In some of the cases which I have observed there was marked wasting with loss of muscular power. One thing characteristic of all these pains is that they are not constant. They come and go and are influenced by weather conditions and barometric changes.

These cases have been misunderstood, wrongly diagnosed and consequently wrongly treated. They are quite often treated for rheumatism.

The following cases will illustrate the severe form:

Mrs. B., aged 32, had suffered from a slight infection of the right apex and an advanced tuberculous process of the upper left lung which had ended in cavity formation and healing. She had also had tuberculous ulceration of the interarytenoid space and left cord which resulted in healing. She suffered a great deal from aching of the left shoulder during her illness, and during the latter part of her treatment suffered severe pain running up the left side of the neck, which persisted, at intervals, for months and then finally disappeared.

Mr. L. consulted me in February, 1911, for a wide-spread ulcerative condition of the larynx. Upon examination of the chest I found an old fibroid lesion of the right lung occupying the upper half of the upper lobe, which was the seat of renewed activity. I told him my findings, that he had evidence of an old lesion in the lung. He protested that it could not be. I inquired as to his reason for leaving Philadelphia, which was his former home. He said that he left be cause he had suffered from a severe rheumatism of the right shoulder which partially incapacitated him for business. I asked if he was examined. He said that he had consulted nearly a dozen of the best men there, all of whom had pronounced the case rheumatism. None had given him an idea that tuberculosis was present. During his last illness, for it proved fatal, he again suffered to some extent from the same old pains. I have no doubt that his pains were due to a neuritis of reflex origin, the cause being the tuberculous process in the lung.

Mr. G. consulted me in January, 1912, suffering from a pain in the upper part of the right chest which at first was considered to be of pleural origin. Owing to my inability to understand the patient's language, I was somewhat deceived for a few days. I found upon examination that the major portion of the upper lobe on the right side was involved in an old chronic tuberculous process and at the apex there were signs of cavity formation. His clinical history was interesting. He had always enjoyed good health and, being a man of means, had lived well. In September, 1911, he was taken suddenly ill with chill followed by fever, severe cough, free expectoration and hemorrhages. Patient made a good recovery and had no further trouble until December, 1911, when the same thing occurred again. This was followed in about two weeks by the pain which I mentioned. The patient was admitted to the sanatorium and carefully watched; the pain persisted, coming on in severe paroxysms two or three times a day, sometimes

requiring morphine. It lasted from a few minutes to one or two hours at a time. It is now nearly six months since it started and it seems to be gradually becoming less severe and less frequent. The pains seem to be confined to the brachial plexus, and particularly certain branches of it: viz, the dorsalis scapulæ supplying the rhomboidei, the thoracales anteriores supplying the pectoralis minor and major, and the axillaris supplying the deltoideus and shoulder joint. Many of the other branches are also involved but these show the greatest pain. The muscles covering the anterior and posterior surface of the upper part of the chest, the shoulder and arm are all markedly degenerated and the strength of the right arm is very much reduced.

It is very necessary to recognize these pains for they offer suggestive diagnostic hints. Any of these pains, especially if they are confined to the shoulder or the upper portion of the chest, call for careful examination of the lungs, to exclude a pulmonary involvement before any other diagnosis is made. The reflex path involved is very plain, as will be seen from Fig. 16 (p. 81), when we remember that the cervical sympathetics supply the lung.

CHAPTER IX

The Effect of Muscle Change Upon Percussion Findings in Physical Examination of the Chest

That the muscles are important factors in the production of the phenomena elicited by percussion of the chest can be easily understood. The difficulty of obtaining information as to the condition of the posterior portion of the apex of the lung in a subject with heavy musculature is appreciated by diagnosticians generally. It is also recognized that percussion over the anterior (except the mammary areas in women), lateral, and posterior inferior portions of the chest is more satisfactory than in other portions.

I would also call attention to the fact that the percussion note is more resonant and the resistance to the finger used as a pleximeter is less over all areas where the musculature is thin than where it is thick.

This is very evident in the axilla. There are other areas also to which I wish to call attention; for the changes in percussion over them lead to erroneous opinions being given. In the first intercostal space anteriorly, at about its middle (see Fig. 2, p. 19), there is an area which is thinly covered by muscle. It lies between the clavicular portion of the pectoral and deltoid muscles. This area is wider in some subjects than in others and the percussion change described varies with the width. In percussing either from the sternum or from the shoulder, a certain note is detected and a certain resistance felt until the middle of the intercostal space is reached when, if this area between the muscles is of some width, an increased resonance and lessened resistance is noted. Other areas showing the same percussion phenomena, and even to a greater degree, are found near the sternal ends of the intercostal spaces. This is due to the fact that this portion of the chest is not so thickly covered with muscle as the outer portion. See Fig. 15. The inner borders of the pectoral muscle arise from the sternum; and, its sternal portion—that is the portion covering the inner third of the intercostal spaces—is thin, while the outer two-thirds is fleshy. Not only is the outer two-thirds fleshy but its thickness is

also reinforced in certain parts by deeper muscles. The percussion findings over these areas covered with little muscle differs so much from that over the fleshy parts that it often gives erronous impressions. This is especially true when the underlying lung is the seat of advanced tuberculosis and the muscles have degenerated. Under such conditions the percussion note is often so changed that it is er-

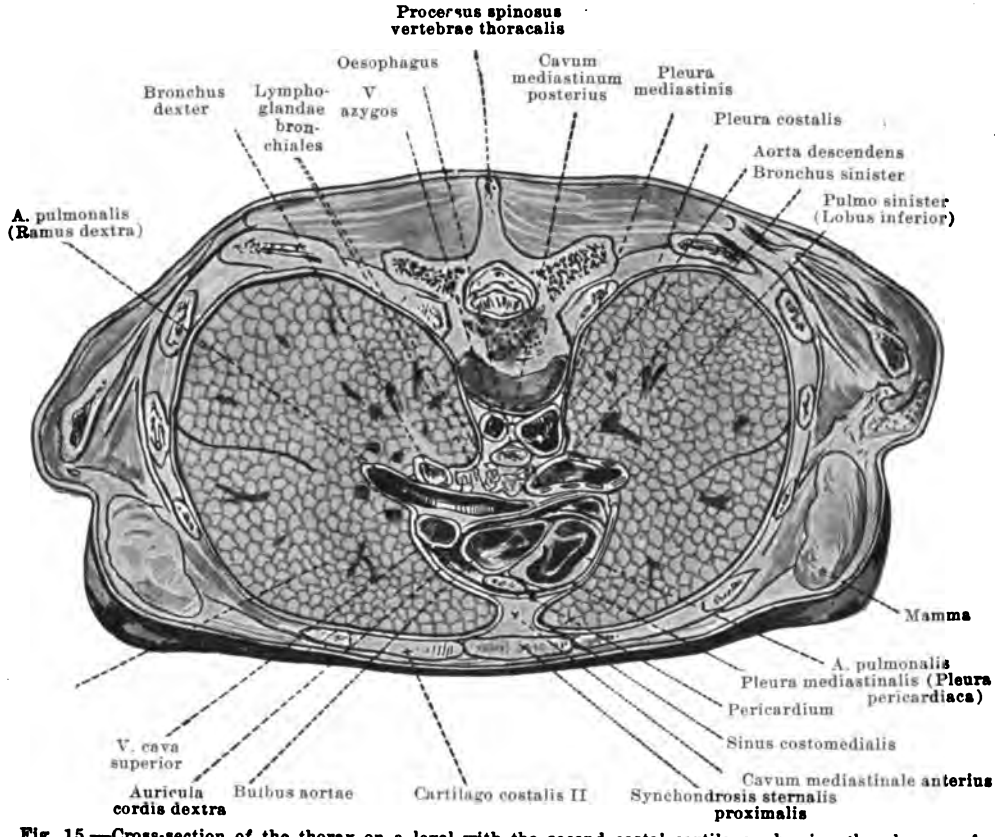

Fig. 15.—Cross-section of the thorax on a level with the second costal cartilage, showing the absence of muscle over the intercartilagenous space. There is no fleshy muscle on the chest wall at this level from the sternum until after the costocartilaginous junction has been passed. This gives different percussion and auscultation findings from that portion further toward the axilla where the muscle is thicker. Illustrations usually show the pectoralis as beginning at the sternum. The portion near the sternum, however, is very thin compared with that portion further toward the axilla, and this difference must be taken into consideration in the interpretation of data obtained on percussion and auscultation, especially when these muscles have degenerated as a result of chronic intrathoracic inflammation.

roneously interpreted to mean, as mentioned in a previous paper (48), either the presence of emphysema or cavity.

While it is important to know these facts pertaining to normal muscle conditions, it is to the effect of the muscle spasm and degeneration upon percussion findings that I wish especially to call attention at this time.

In order to obtain an idea of the effect produced by contraction and relaxation of muscles upon the percussion findings in physical examination, I would suggest that the examiner compare the percussion findings over some muscle such as the biceps during relaxation and contraction. It will be seen that contraction gives an increased density to the muscle tissue which produces increased resistance to the finger and a note of higher pitch.

What is true of the biceps is true of the muscles in general. We note the same changes over the muscles covering the chest as can readily be determined by percussing over the pectoral muscle in a healthy subject while the muscle is relaxed and comparing it with the findings when the arm is fixed so as to throw the same muscle into a state of contraction. This can be shown by any other muscle or sets of muscles that have sufficient mass and are able to be thrown into a state of contraction and relaxation at will, such as the neck muscles, trapezius, and rhomboidei. When one perceives the change on percussion over the muscle when it is relaxed as compared with when it is contracted over healthy chests, he is prepared to recognize that the same holds true over chests the seat of pathological change.

Recognizing, then, that the muscles of the neck and chest are thrown into spasm by acute inflammatory conditions of the underlying pulmonary parenchyma, we must see that we have the same percussion phenomena produced by the muscle change as is produced by, and usually described as being due to, the infiltration and other inflammatory changes in the lung. This change in the percussion findings which is due to the contraction of the muscles is of special significance in those instances where the findings are not far from the normal, as is usual in early tuberculosis. The infiltration is not dense enough to cause marked percussion change of its own accord; but the muscles being tense from spasm, emphasize the percussion findings. In slight inflammatory infiltration the muscles are probably the largest element in percussion change. When the disease becomes more marked, however, the infiltration being greater or scar tissue becoming more predominant, as in advanced tuberculosis, pneumonia, lung abscess, lung syphilis, actinomycosis, cancer, and mediastinal tumors, the muscle spasm ceases to be so large a factor in the percussion findings and becomes secondary to the density of the lung and neighboring tissues. When the process has existed for some time and the muscles have degenerated, this must be taken into consideration in interpreting per-

cussion findings. I have seen the pectoralis, trapezius, and levator anguli scapulæ so degenerated over a lung that was the seat of a healed tuberculous process, that the percussion note over the other lung was relatively so much higher in pitch and offered so much greater resist-

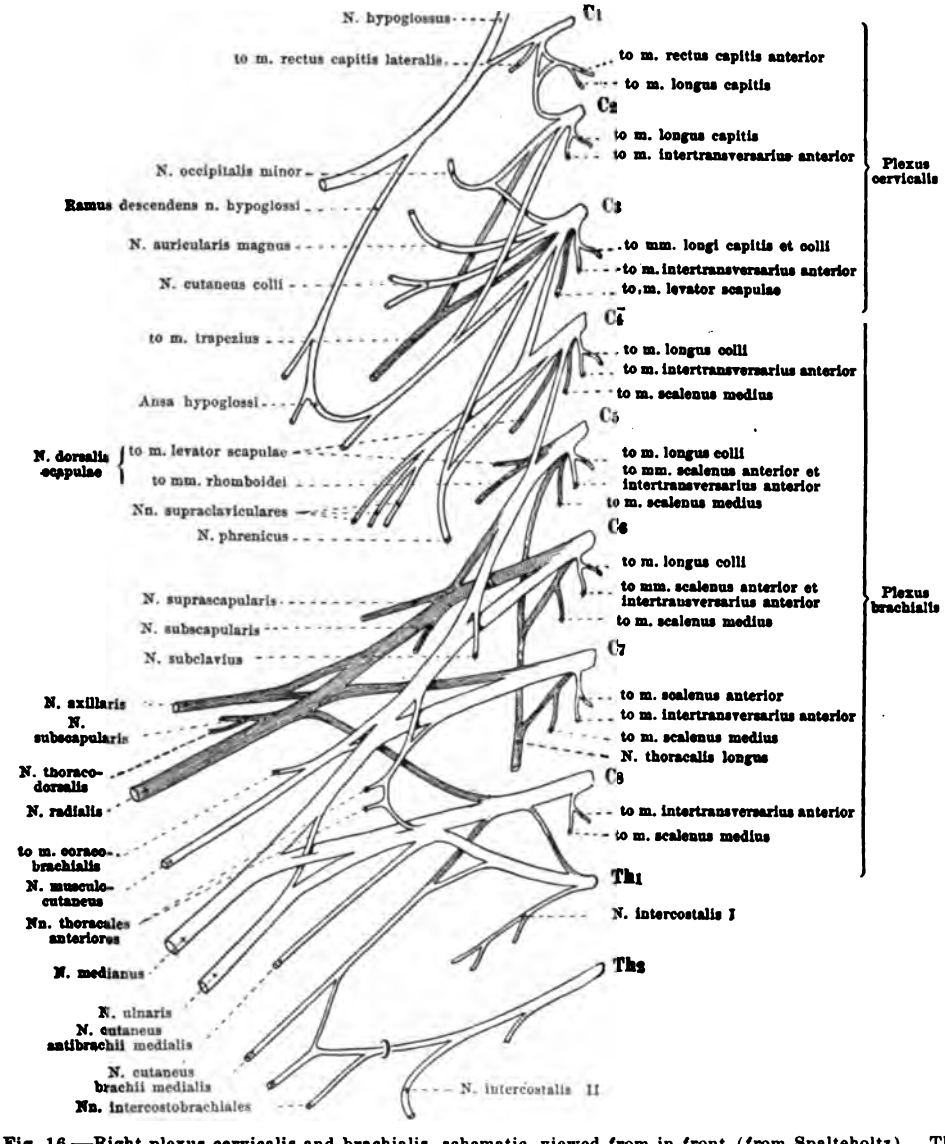

Fig. 16.—Right plexus cervicalis and brachialis, schematic, viewed from in front (from Spalteholtz). The darkly shaded strands are derivatives of the dorsal half of the plexus.)

ance to the finger that I should have felt sure that it was diseased had I not been aware of the effect of muscle conditions upon the percussion note.

A failure to recognize the degeneration of the muscles and the loss of subcutaneous tissue over an apex which has been the seat of a chronic or quiescent lesion will often cause false interpretations of percussion findings, as can readily be understood from the following observations.

Case I: Male, aged 28.—Three years previous to present illness had a period of ill health, felt tired, lost weight, had chronic cold, with slight cough and scant expectoration. Changed occupation, after resting for a time, and apparently regained health. Recently, patient showed similar symptoms again. On examining the patient stripped, the following interesting conditions presented themselves. The muscles and subcutaneous tissues over the right apex were degenerated. The degeneration extended to the third rib anteriorly and to the spine of the scapula posteriorly. The muscles over the left apex appeared normal. On percussion the note over the left apex was considerably higher in pitch and offered considerably more resistance to the palpating finger than that on the right. From this, according to our text books, we should have expected an infiltration of the left apex, but further examination showed this to be improbable. Auscultation of the right apex showed some roughening of the respiratory note which was very harsh on inspiration and markedly prolonged on expiration, while the sounds over the left apex were apparently normal. Here the data obtained on percussion pointed to a lesion at the left, while auscultation, inspection, and palpation showed a lesion at the right apex. The loss of the muscular substance and the subcutaneous tissue was more than sufficient to overbalance the extra tissue produced by the thickening of the lung substance, consequently the normal tissues on the left gave a higher percussion note.

Case II: Female, aged 21.—Came for examination complaining of symptoms indicative of early tuberculosis, malaise, lack of endurance, loss of weight, slight hacking cough and a history of having spit up some blood. Physical examination revealed slight wasting of the subcutaneous tissue and degeneration of the muscles about the left apex, especially posteriorly. The suprascapular fossa was flattened and the supraclavicular notch was deepened. On the right, however, the shoulders appeared somewhat fuller than normal, the suprascapular fossa being distinctly rounded. On percussion the pitch of the note was about equal on the two sides. From our text books we would be led to think of a lesion at the left apex because of the wasting, giving

us an idea of a retracted apex and also because the percussion note approached that of the right in pitch. Palpation revealed a distinct resistance (spasm) on the part of the shoulder muscle on the right, while those at the left were degenerated but not in spasm. Auscultation showed harsh inspiration with harsh prolonged expiration over the left apex, and roughened inspiration, prolonged expiration, and impeded breathing over the right apex. The latter accompanied by a few fine rales on coughing. The indication from percussion would have been a lesion at the left apex. The diagnosis from the condition of the muscles as determined both by inspection and palpation was an old quiescent focus at the left apex and a new active lesion at the right apex. Auscultation confirmed the latter.

In both of these cases the results of percussion were misleading. I am seeing cases almost daily where the condition of the muscles, taken into consideration with my percussion findings, are of the utmost value in interpreting my findings.

This suggests to the examiner the necessity of having all muscles either in a state of relaxation or contraction, if comparative observations are to be made, and shows that exactness in percussion demands that the condition of the muscles be carefully considered. It shows, as mentioned above, that the method which is often employed of percussing one apex with the face forward and the neck muscles relaxed and the other with the face turned to the opposite side making the neck muscles tense, produces data which is unsuitable for comparison.

Fig. 16, p. 81, shows the nerves through which the impulses, which cause the spasm and degeneration of the muscles, pass.

CHAPTER X

The Effect of Muscle Change Upon the Data Derived From Auscultation

The effect of the muscles upon the respiratory murmur has been recognized by clinicians unknowingly. Teachers of physical diagnosis teach their students that if they will fix in their minds the normal vesicular murmur, they should listen to the respiratory murmur in the axilla. The facts are that the respiratory note heard in the axilla cannot be expected to be heard over the front or back of the chest. Among other reasons why the murmur in the axilla is soft and breezy is because it is not modified by thick masses of muscle. What would be considered normal vesicular breathing for the axilla, however, would not be normal vesicular for the apex, the pectoral or interscapular regions, where the murmur is changed by the muscle masses through which the sound is heard.

The normal respiratory sounds differ, then, according to the tissues through which they pass, as well as according to the condition in which the tissues themselves are found. This will be readily appreciated when we discuss the difference in respiration when the muscles are normal and when in contraction.

The areas which I mentioned above as showing lessened resistance and greater resonance on percussion also show a respiratory murmur which is more distinct and softer than the areas covered with thicker muscles.

The same experiment with the pectoral muscle over a healthy chest that I mentioned in discussing percussion may be carried out with auscultation. If one wishes to convince himself that there is a large muscular element in our auscultatory findings, let him listen over any of the important muscles of the body and compare the sounds heard during easy and forced contractions. Then, in order to impress him more thoroughly, let him listen to a healthy lung over the pectoral muscle, first, when the muscle is relaxed and then when it is contracted. The sound heard over the relaxed muscle is a soft breezy murmur, not so soft, however, as that found where the musculature is thin. That

heard over the muscle when contracted, on the other hand, is very different and appears as slightly roughened, harsher than normal, with a prolongation of the expiratory sound. The murmur sometimes seems much weakened. These phenomena are well described by the term "impeded breathing," which I have applied to the early signs so often heard in incipient tuberculosis of the apex.

These experiments, together with auscultation over portions of the chest covered by little and much muscle, will show that the muscles are important factors in the causation of the respiratory murmur, and will prepare the way for the statement that the muscular element is a very important one in the production of certain changes in the respiratory sounds in diseases within the chest.

The muscular element is greater in those diseases such as early tuberculosis, when the changes in the lung itself are slight and less when the changes in the lung itself are more pronounced, the same as mentioned above in our discussion of percussion. In early apical tuberculosis the sounds heard on auscultation are very largely those that we hear in a normal chest over the pectoralis when in a state of contraction. This gives weight to the opinion that the muscles cause much of this altered sound.

The muscles covering the apex being reflexly thrown into a state of tonic contraction by the underlying inflamed lung, afford the conditions which simulate those of the pectoralis when artificially contracted, and, what is to the point, are accompanied by the same auscultatory phenomena that are heard over the pectoralis when in a state of voluntary contraction in a healthy chest.

I do not wish to convey the idea that the respiratory sound originates wholly or largely in the muscles. What I wish to emphasize is that a part of it originates in the muscles and that that which does not originate there is modified by them. Thus, where there is little change in the respiratory note, but great change in the muscles, as we find in small apical pulmonary lesions, I am inclined to believe that the alteration in the respiratory note may be largely affected by the muscles; but in conditions when the pathological change in the pulmonary parenchyma is extensive, the effect of the muscles upon the sound is inconsequential.

CHAPTER XI

Light Touch Palpation

A DISCUSSION OF THE POSSIBILITY AND PRACTICABILITY OF DELIMITING NORMAL ORGANS AND OF DIAGNOSTICATING DISEASED CONDITIONS IN ORGANS, ESPECIALLY WITHIN THE CHEST AND ABDOMEN, BY VERY LIGHT PALPATION WHEN SUCH NORMAL ORGANS AND DISEASED CONDITIONS EXHIBIT A DENSITY OF TISSUE DIFFERING FROM NEIGHBORING ORGANS OR PARTS OF ORGANS.

While making a clinical study of the muscle changes previously described, I noted that these changes were best detected by a very light touch. Further study showed that by this very light touch the examiner was not only able to determine differences in the superficial tissues, but, to my great surprise, he was also able to detect the differences in density of tissues which were situated within the great cavities of the body at a distance from the surface walls (20, 21, and 22). The heart, liver, stomach (within two or three hours after a meal and even without food, provided it contains some gas), abdominal tumors, mediastinal tumors, infiltrations of the lung (whether due to tuberculosis, pneumonia, actinomycosis, syphilis, or cancer), and effusions either pleural or peritoneal can be accurately outlined by *light touch palpation*, a touch so light that it scarcely causes an indentation of the superficial tissues.

The boundaries of the heart were the ones that I detected first. I found that by simple touch I could distinguish the outline of this organ from the surrounding tissues. Inasmuch as I was studying the reflex contraction of the muscles at the time, I at first thought this must be a skin reflex, due to the contraction of the erectores pili, and so described it as a new physical sign, probably a skin reflex; but, before my paper was published, I had determined that it was not a reflex at all, consequently not a new physical sign, only a new method of applying palpation (21).

Many doubts arose in my mind from the first as to whether or not it was a reflex, for as I said in my first paper: "One thing that is difficult to understand is that this sign shifts with the organ. We might

conceive of a certain connection between the skin immediately overlying an organ and the organ itself; but to explain shifting of the sign to other areas of the skin when the position of the organs changes, is difficult. When the heart shifts, as it does in diseases of the lungs and pleura, the sign shifts with it. I have found it to follow the heart, when the organ was entirely to the left of the sternum, and also, in a case of dextrocardia, when the entire organ was to the right of the sternum. I have also found it to follow the liver when it was three finger breadths below the costal border."

Shortly after writing the above I had access to the large post mortem material of the Pathological Institute in Vienna and found that I could outline the organs on the cadaver, though not as readily as on the living subject, the difference being probably due to the stiffening of the tissues after death. This eliminated the idea of a reflex and proved that what I had really observed was the fact that it was possible to recognize the difference between organs or parts of organs of different density by simple touch, even though lying within the large cavities of the body. The fact that by a touch so light that it scarcely indents the skin, one can outline organs whose borders are at a distance from the surface of the body, is of great import in further developing physical diagnosis. This calls for a new consideration of palpation and percussion as diagnostic methods and especially requires a thorough, careful discussion of the relative merits of light and heavy percussion and also of the feeling of resistance on percussion in contradistinction to the note elicited by the stroke.

The data obtained by percussion depends upon the fact that when a stroke is delivered upon the surface of the body in immediate percussion, or upon some intervening object used as a pleximeter in mediate percussion, vibrations are produced and the tissues are disturbed. The impact sets up vibrations which penetrate the tissues and emit sounds and produce varying feelings of resistance. The character of the sound and the nature of the resistance depends upon the manner in which the vibrations are interfered with by the tissues through which they pass. For many years the sound emitted by the percussion stroke was made the basis of judgment; but, in recent years, the resistance to the finger has rapidly assumed greater importance. With this, percussion becomes a method of palpating. If we take as the basis of our judgment the resistance felt on percussion, then, percussion and palpation are based on the same principles: first, the sense of

touch or perception which is found in its highest state of refinement in the pulps of the fingers; and, second, on the manner in which the vibrations which are induced when the fingers come in contact with the objects in palpation and percussion are interfered with by the various tissues. I presume that no one will question the first of these principles, for if it were not for this touch sense there would be no perception. The second principle, however, which seems equally self-evident when fully considered, may not be so readily accepted; but, a little thought will remove all skepticism; for, the difference in impact between the skin and the finger which is gently laid upon it, as it is in palpation, and, when it is forcibly driven against it, as it is in percussion, is merely a matter of degree. Palpation furnishes data by a light stroke and light touch palpation by an exceedingly light stroke, while percussion furnishes it by a heavier stroke. Runeberg (49) in discussing one of my papers, takes the same view.

The results of our studies show that we feel much deeper than we have been wont to think. We have been in the habit of looking upon light palpation as a method suited to examining the surface only and have thought of it as depending entirely on the sense of touch; but we are now convinced that even a very light touch sets up vibrations in the tissues which not only penetrate deeply, but which are able to penetrate several different tissues of different density. A splendid illustration of this is the outlining of the deep borders of the heart. The left edge of this organ in muscular subjects may be in the neighborhood of two inches from the surface; and the vibrations are compelled to pass through the skin, subcutaneous tissue, muscles, ribs, pleura, lung, and pericardium (and, in women, the breast) before they reach the heart. In their passage, not counting the skin, pleura, and pericardium, there are three or four tissues according to whether we palpate over the intercostal space or the rib, each of different density and each of considerable thickness, through which the vibrations must pass. That such a method could be reliable seems incredible, yet we have checked it by all forms of percussion and proved it with the orthodiagraph as will be mentioned later, and found that it can be depended upon to furnish reliable data.

Physicians who are expert in physical diagnosis have been rapidly discarding heavy percussion because of its unreliability as well as because of the discomfort which it often causes the patient. A percussion stroke sets up vibrations not only at the point of the stroke, but

also for considerable distance around. The heavier the stroke the more evident this is. If a heavy stroke be delivered over the bony thorax, it sets the entire thorax into vibration and confuses the examiner. Often, small areas of increased density in the lungs are overlooked when heavy percussion is employed. When a light stroke is employed, on the other hand, the maximum effect is produced at the point of, and in the direction of, the stroke with a minimum of extra and confusing vibrations.

Not only is heavy stroke percussion giving way to light stroke; but, for outlining solid organs, infiltrations and hollow organs while filled or partially filled with liquids or solids, as well as determining alteration in the tension of air containing viscera, the sense of resistance felt on percussion is rapidly assuming an equal if not a greater importance than the sound emitted by the stroke. Now, that we have determined that a very light touch sets up vibrations which reach the deeper organs and that their penetration of these organs can be perceived by the palpating fingers, we have stronger reasons for developing the light stroke. Heretofore, it has been argued by many, who were committed to the light stroke for general percussion, that a heavy stroke was necessary for the examination of organs or parts at a distance from the surface. We have proven this to be untrue by outlining the deep borders of the heart anteriorly as well as posteriorly and also mediastinal tumors (enlarged tuberculous glands and carcinoma) by touch.

In employing *light touch palpation* considerable care is necessary at first, until the examiner is fully aware of what he is attempting to feel. He should remember that he is attempting to recognize slight differences. In outlining organs or differences in density of tissues, I have found it better to examine the part which is presumably of lesser density first, and pass from it to the organ or part of greater density.

One thing that surprised me very much was the fact that *light touch palpation* could be relied upon to give accurate results through bone as well as soft tissues. This fact led me to make further experiments. While studying percussion I practiced upon tables and chairs, locating the legs and reinforcing strips; so I tried to do the same by palpation and found that it is possible to note a difference in resistance on palpation also when palpating over the chair and table legs and those parts which are reinforced. While this is possible, it is not easy to do, and no one need feel discouraged if he finds that he is unable to do it

at the first trial. I do not mention this as a practical method but simply as a scientific fact bearing upon the question under discussion. For those who doubt that touch will set up vibrations which can be made to pass through solid substances in such a manner that they may be perceived, I would cite the recent photographing of the heart sounds. These sounds are transmitted through a wooden pole, the sound waves are then given off into a rubber tubing and conveyed to an electrical apparatus by means of which they are reproduced upon sensitized paper. If sounds will set up waves, which, after passing through wood, may be collected and reproduced photographically, it is no stretch of the reasoning power to believe that the vibrations set up by touch will also pass through solid substances.

Bearing upon the point of being able to differentiate objects of different density by touch, I would also like to call attention to the fact that the blind are taught to distinguish by touch the difference between brass and gold.

For one that is trying light touch palpation for the first time, I would recommend that he try and outline the left border of the heart in a normal chest in a person with a moderate amount of subcutaneous tissue. The palpation should be done with the pulps of the fingers where the sense of touch is developed the keenest. The resistance over the heart is best felt over the area of absolute dullness, therefore I would suggest that the resistance over the fourth interspace about one inch to the left of the sternum be noticed and compared with that in the same interspace beyond the edge of the heart. A little careful observation will enable the examiner to see that the resistance is much greater over the heart than it is over the lung tissue. When this difference has been fully comprehended he may attempt to find the border of the heart. He must remember, however, that the border being deeper in the chest and covered with an extra layer of tissue (the lung) will not be as easy of detection as the heart over the area of absolute dullness. The resistance felt will be less. As mentioned above, the method which I like best is to begin beyond the heart border and then gradually approach it, carefully watching for the least sign of increased resistance. Both borders of the heart should be approached systematically in each intercostal space or, if the examiner prefers, over each rib. For beginners I would recommend the intercostal spaces.

Quoting from one of my former papers (50):—

"I have found the best method of procedure in endeavoring to outline the deeper solid organs by *light touch palpation* to be as follows:—

"1. Always palpate wholly, either in the intercostal spaces or over the ribs. This can be applied to the liver and spleen, as well as the heart, because of the oblique direction of the ribs.

"2. Begin palpating beyond the border of the organ and approach it slowly. When the border is reached an increased resistance is at once noted, the degree varying in different chests.

"3. The palpating fingers must not be moved too rapidly, or confusion will result. Sufficient time must be allowed to concentrate the mind on the sensation produced at each touch. Concentration is very important, especially when the change is slight, as would be noted in a heart or liver border covered by emphysematous lung in a patient with thick chest walls."

The next question for discussion is the use to which light touch palpation can be put. In examining the chest, aside from the usual use made of palpation, it may be used for practically every purpose for which percussion, depending upon resistance, is used.

I have already described its use in outlining the heart but would like to add that it can be employed very successfully in determining the lower border of the heart as it rests upon the diaphragm. This is of special interest, because it is differentiating by touch, the heart from the liver. If one doubts this, let him, after he has become proficient through practice, begin slightly to the left of the medium line below the ensiform cartilage and then slowly approach the heart, palpating the tissues carefully. He will notice a distinct change in resistance when the lower border of the heart has been reached. In this procedure it is best to follow the costal arches palpating over the cartilages.

The liver offers several interesting problems in palpation. In the first place, the liver changes its position at each respiration, consequently it must be examined while the patient holds his breath or while he breathes quietly. The upper border is somewhat difficult, because in the first place, it is at a distance from the chest wall, and in the second place, because in approaching it from above anteriorly, it is necessary to cross the ribs, going from interspace to rib. In a normal chest, however, there is no difficulty in differentiating the absolute dullness from the lung tissue above and practice will soon show that

the upper border of the liver may be outlined accurately, regardless of the difficulty mentioned. In the axilla it is much easier because of the oblique position of the ribs. Beginning high up in the axilla above the liver, palpating either in the intercostal space or over the rib, the examiner should gradually approach the liver. When its upper border is reached an increased resistance will be noted which still increases as the area of absolute dullness comes under the palpating finger. The lower border is sometimes easily determined, at other times it is determined only with difficulty. To facilitate palpation it is necessary to reduce abdominal breathing to a minimum; then, beginning a few inches below the border of the ribs one should gradually approach the organ until its presence is determined by a varying degree of resistance according to the consistency of the liver tissue and the thickness of the belly wall. The left border of the liver is to me extremely easy to find on palpation, as a rule. If one will begin beyond the nipple line, and following the ribs gradually approach the median line he will note in a normal subject a decidedly increased density when the area corresponding to a perpendicular line through the apex of the heart has been reached, which he will find corresponds to and is the left border of the liver.

One of the most interesting facts connected with palpation is that the stomach can be outlined by it. This is outlined easiest soon after a meal, but I have been able to do it successfully three and four hours after a meal in some subjects. The upper border can be outlined readily by the lack of resistance when the organ is filled with gas. At other times, its location would be extremely uncertain. The lower border can, as a rule, be easily differentiated from the intestines by an increased resistance within two or three hours after a meal, the left border is usually easily made out during this time also and at times the pyloric end can be followed out across the median line, Another very interesting fact is that the stomach may be outlined by palpation when it is practically free from food, provided it contains a little gas, sufficient to separate its walls slightly. This condition gives a very peculiar sensation to the palpating finger which will not be forgotten when once determined.

The spleen can be outlined by palpation and when enlarged this can be done with ease.

Not only is light touch palpation of value in outlining normal organs but it is also of value in pathological conditions.

Infiltration in the lung, anything making the tissue dense, can be noted. Of course the pathological process must be sufficient to cause an appreciable change from the normal. Tuberculous infiltrations and resulting scar tissue can readily be determined by increased resistance felt on palpation. Pneumonias are easily detected. In lobar pneumonia I have been able to determine the division between the lobes by palpation as accurately as though the lungs were exposed. The presence of cancer, syphilis, actinomycosis, lung abscess, in fact all diseases which change the density of the pulmonary tissue, can be detected by palpation. In tuberculous infiltrations of the lungs it is my every day practice to outline the infiltrations and consolidations by palpation, noting the difference in the density of the tissues as they are perceived by the touch. This is distinct from the spasm of the muscles, although it is easily confused with it. At times it is difficult to tell how much of the resistance is due to infiltrations, and how much to muscle tension.

Mediastinal tumors, especially if of any considerable size, are also quite satisfactorily outlined by light touch.

I would also like to call attention to the value of palpation in conditions where the density of the tissue is lessened, as pneumothorax, and where there is a loss of tissue as in cavities in advanced tuberculosis. By palpating over cavities we can often note the lack of resistance to the finger as compared to the tissues beyond. Where the chest wall is not too thick, palpation will often suggest the presence of cavity; and I might add, that, to my mind, there is probably no sign of cavity that is more constant than this lack of resistance on palpation and palpatory percussion. The value of palpation in cavity is that this lack of resistance can be detected when the usually described signs, such as blowing expiration, amphoric breathing, pot fêle, and Wintrich's sign are not to be found. These latter signs all depend upon the presence of certain physical conditions in the cavity for their production, hence are extremely variable in their appearance. The lack of resistance which I mention simply depends upon the absence of tissue and should always show unless the cavity is filled with secretion, and if the cavity is surrounded by a dense wall the line of demarcation is easily felt. The resistance afforded by thickened pleura and pleural effusion are also readily determined by palpation.

Emphysema, especially if very marked, presents rather a peculiar

sensation to the palpating finger. The ribs are forced outward and the intercostal muscles are put on a stretch so that they offer a resistance not unlike that presented when the same muscles are thrown into spasm by acute pleurisy. This is often quite confusing in patients suffering from advanced pulmonary tuberculosis where pleurisies and compensatory emphysemas are apt to be present. Points of value in differential diagnosis, aside from those found on auscultation, are the pain if the pleurisy is acute, and whether acute or chronic, the lagging present in pleurisy and the lack of it, or even bulging in emphysema.

The lower border of the lungs can be determined very readily by palpation. Anteriorly, following down the oblique intercostal spaces, the palpating finger notes a very different sensation when it leaves the border of the lung and comes in contact with the tissues devoid of air. The same is true posteriorly. From the air-containing lung tissue to the non air-containing tissues, the line of separation is not difficult to determine. Practice makes all these procedures easier, but when this difference is once perceived it is not difficult to repeat. I have outlined the apex of the lung by touch and done it as accurately as by the method of either Goldscheider or Krönig.

In all acute inflammatory conditions, affecting either the pulmonary parenchyma, or the pleura, we are somewhat at a loss to explain how much of the resistance on palpation is due to the spasm of the muscles covering the thorax and how much to the increased density of the tissues (or the fluid in pleural effusion). In slight infiltrations and in acute pleurisy it is of course nearly all due to the muscle rigidity; but when the infiltration of the tissues is greater, or when the pleura becomes thickened or an effusion takes place, then the muscles are of secondary importance to the tissues or fluid in the production of resistance.

Much suggestive information can be obtained by examining the muscles of the thorax and abdomen by *light touch palpation.* The muscle changes, both spasm and degeneration, may readily be determined in this manner; and, when found, are indicative of either an acute, a chronic, or a previously existing inflammation. These muscle conditions as they appear in diseases of the thorax I have already described. The changes in the abdominal muscles can also be detected by *light touch palpation.* I feel sure that many inflammatory conditions of the abdominal viscera are reflected in the abdominal muscles

but overlooked because the examiner palpates too heavily. Heavy palpation blunts the sensibilities somewhat; so it seems to me that, at least for palpating superficial tissues such as the muscles, the pressure should be very light. If the examiner will systematically examine the musculature of the abdomen by *light touch palpation* he will be surprised to find that he often has hints of underlying visceral diseases given to him by the rigidity of the musculature which would have been entirely overlooked by the usual heavy palpation.

As an illustration of the superior value of *light touch palpation* in the diagnosis of abdominal tumors, I will cite the following case. Child, aged 14 months, was taken suddenly ill with restlessness and fever of 104. Examination of the mouth revealed the fact that the child was cutting four teeth. This was taken for the cause of the trouble and a restriction of food and castor oil was ordered. Thirty-six hours later the child passed several small stools containing mostly mucus, but no blood. Fifty hours after the onset the child vomited fecal matter. During this entire time the temperature remained between 104 and 105½. Six hours after the vomiting began I first saw the child. Temperature had fallen to 102 and the child was in a state of collapse. Examination showed the abdomen distended. On *light touch palpation* I outlined a tumor below the umbilicus immediately to the left of the median line. While all of the abdominal muscles were tense, the left rectus, immediately over the tumor, was extremely so. My assistant examined the patient also and by *light touch palpation* made out the same tumor. Another physician and another surgeon were called in who examined the child by the usual heavy palpation. They also examined the case per rectum, but failed to find the mass. However, they noted that the left rectus was more rigid than the right. The child died on the seventh day and post mortem revealed a perforated appendix. The appendix was lying below the umbilicus and to the left of the median line, lying against the sigmoid flexure where we had located the tumor by *light touch palpation.* Around the appendix there were several ounces of pus. The greater rigidity of the left rectus in this case can be explained by the localized peritonitis present, part of the resistance, however, determined by palpation was due to the appendiceal tumor.

My study of muscle reflexes would indicate that a visceral inflammation should always reflect in the same muscles no matter where the viscus is situated. This offers a valuable suggestion for differ-

entiating appendicitis from other acute visceral inflammations such as those of the gall-bladder and ovary. No matter whether the appendix be in its normal position or down over the brim of the pelvis, or up under the edge of the ribs, or over on the left side, the muscle spasm should be in the same classic place, for, no matter where the organ is, its nerve supply is the same. Of course if peritonitis were present, the greatest spasm would be over the inflammation of the peritoneum. Its detection should be facilitated by light touch. Heavy touch confuses.

Abdominal tumors and pelvic tumors, when they reach the abdominal cavity, can at times be accurately outlined by light touch. It is surprising to see how these dense masses, even when not adherent to the belly wall, may, at times, be detected. Peritoneal effusion is readily found by *light touch palpation*, the level of the fluid offering a line of demarcation which is recognized at once by the difference in resistance.

In two cases of tuberculosis of the kidney, one unilateral and the other bilateral, where diagnosis was confirmed by operation in one case and by segregation of the urine and animal inoculation in the other, I was able to detect marked rigidity of the lumbar muscles by *light touch palpation*. In the bilateral case, the left kidney lost all signs of disease and the left lumbar muscles lost their rigidity on palpation. Light touch showed these changes best.

We have now mentioned briefly some of the organs and pathological conditions that may be outlined and diagnosticated by *light touch palpation*. But the question must arise at once to the scientific mind, are the findings reliable? In answer to this question we believe that we can safely say that they are as accurate as those of percussion. I have put this method to very severe tests, comparing it with mediate and immediate percussion, Epstein's touch percussion, auscultatory percussion, and, in case of the heart, with the orthodiagraph, and am pleased to find that its accuracy has been fully established.

I doubted the accuracy of the results obtained by *light touch palpation* when I first discovered it; in fact, I could not believe that it was possible to feel through tissues with so light a touch, and when I found its results corresponded with those obtained by percussion, I was amazed, but I still felt anxious to see it tested with methods whose accuracy could not be questioned. While in Vienna, in 1909, the opportunity offered to test this method and control it by the ortho-

diagraph. I first marked out the borders of the heart by *light touch palpation.* Then Dr. Schwartz, assistant in the Holzknecht X-ray Clinic, outlined it by the orthodiagraph. The comparative results based on the examination of fifteen hearts in normal chests as published in a previous paper (20) were as follows: "In nine or 60 per cent. of the cases both borders coincided, in 12 or 80 per cent. the right border coincided, and in 12 or 80 per cent. the left border coincided. In no instance was the error more than 1½ centimeters. Of the three right borders that I missed by *light touch palpation,* two were among the first five patients that I examined.

In approaching the borders of the heart, especially the right, on percussion, if one will give close attention, he will note a resistance to the finger just before the real border is reached. The same may be noted on palpation and was the cause of my error.

The accuracy of this method, like all others, depends upon the examiner. Practice will convince one of its value, and is necessary to good results. It is not as difficult as it might seem on first thought. I have demonstrated it to many men who have taken it up at once and applied it to their daily practice. It is a great advantage to have many methods for they correct each other. No one can deny the advantage which arises from being able to quickly place the hand on the chest and say "there is the border of the heart," or "there is the upper border of the liver," "here is a pulmonary infiltration, or probably a thickened pleura."

Extremely thick chest or belly walls, large mammary glands, and extreme grades of emphysema all interfere with the ease of examination, but, to my mind, they do not alter its accuracy any more than they do the accuracy of percussion.

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